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The Journal of Nervous and Mental Disease

Vol. 123, No. 6 JUNE 1956 Serial No. 894

CURRENT PATHWAY AND NEUROPHYSIOLOGIC EFFECTS OF ELECTRICALLY INDUCED CONVULSIONS[1,2]

R. B. AIRD, M.D., L. A. STRAIT, PH.D., J. W. PACE, M.D., M. K. HRENOFF, B.S., AND SYLVIA C. BOWDITCH, B.A.

Several studies, concerned with the cerebral pathway of electroshock current, have been conducted in the belief that such knowledge might contribute to an understanding of the underlying neurophysiology of electroshock as used therapeutically in certain psychiatric conditions. Neuropathologic investigations (7, 8, 9, 10, 12, 15, 17, 18, 21, 23, 24) have shown vascular and perivascular changes with currents stronger than those used in human electroshock treatment and again have shown wedge-shaped cortical areas of vasospastic or vasoparalytic effects corresponding to the anatomical distribution of the larger cortical arterioles as they penetrate the cortex from the arachnoid.

A recent study in our own laboratory (6), concerned with the effect of electrically induced convulsions upon the permeability of the cerebrovascular system, likewise has suggested that a considerable portion of the current traversing the brain in electroshock therapy may be transmitted along the vascular tree of the brain or in its blood stream. A marked and relatively persistent increase in the permeability of the cerebrovascular system was produced by a series of electrically induced convulsions in this study.

Considering the anatomy of the vascular tree of the brain and what is known concerning the transmission of electrical currents through tissues, one might postulate that a high percentage of the current traversing the brain in electroshock therapy would concentrate on and be transmitted along the vascular tree of the brain or in its blood stream. According to this concept, only as the current traversed the cortex in the process of its concentration upon the cerebrovascular tree, escaped into the deep subcortical portions of the brain from the deeper blood vessels, or followed neuronal rather than vascular pathways, would the brain be stimulated. With the technique of electroshock therapy in common use, one would not expect this to be a damaging stimulation and presumably might be expressed in the usual neurophysiological terms of neuronal discharge and recovery. At least, one might assume that this phenomenon would not be markedly different from, or worse in its effects than, the changes associated with a "spontaneous" seizure in a clinical convulsive disorder where an abnormal neurophysiological and biochemical state already may be presumed to exist. If this concept is correct, maximal changes might be expected in the vascular tree. No unusual effects over and above those transiently occurring in clinical epilepsy or, at the worst, only minimal changes would be expected in the brain itself.

The possibility of testing this thesis of cerebrovascular transmission appeared feasible by using the techniques developed in our laboratory. In previous studies (1, 3, 4, 5, 27) direct and accurate methods for the extraction, identification, and quantitative determinations of cocaine in brain, blood, and cerebrospinal fluid have been utilized in the study of the permeability of the blood-brain barrier in various conditions. In addition, with the use of the supravital dye, trypan red, which

[1] From the Department of Neurology and the Spectrographic Laboratory, University of California School of Medicine, San Francisco, California.

[2] The work described in this paper was done in part on a grant from the Christine Breon Research Fund and in part on a grant from the Mother Lode Fund.

uniquely stains the endothelial cells of the cerebrovascular tree but which does not pass beyond the blood vessels to gain entrance into the nerve tissue proper, we have been able to lower the permeability of the blood-brain barrier to cocaine and to prevent abnormal increases in the permeability of this barrier, such as occur in many abnormal conditions involving the central nervous system.

These studies have suggested that the locus of the blood-brain barrier[3] resides, as postulated by Spatz (26), in the endothelial cells of the cerebrovascular tree. Furthermore, in our studies on cerebral concussion, an apparent relationship was established between the altered permeability of the blood-brain barrier and the cerebral dysrhythmia associated with concussion. The supravital dye was found to counteract the effect of concussion on both the permeability of the blood-brain barrier and the cerebral dysrhythmia. This had suggested that the dysrhythmia was neurophysiologically related to and depended upon the altered blood-brain barrier effect. Stated in another way, this observation confirmed what was already suspected, namely that the blood-brain barrier is a basic neurophysiologic mechanism capable of conditioning the neurophysiology of the central nervous system. On this basis, the generalized cerebral dysrhythmia associated with concussion could be attributed to abnormal increases in the permeability of the barrier.

If this concept is correct, its application to our previous observation on the increased permeability found after a series of electrically induced convulsions would suggest that trypan red injections prior to such treatment should prevent both the increased permeability and the cerebral dysrhythmia associated with electrically induced convulsions. This, however, would only be true if the transmission of electric current was confined primarily to the cerebrovascular tree, as suggested above. If, on the other hand, the transmission of electric current occurred primarily through cerebral nerve tissues *per se*, as has been postulated by some investigators (7, 11, 14, 20, 25), and if the effects of electroshock were of neurogenic origin, it would seem unlikely that trypan red would abolish the cerebral dysrhythmia usually associated with electroshock therapy. The determination of this point appeared crucial to the question of current pathway, at least from the standpoint of the current producing residual neurophysiologic changes that might be correlated with the relatively persistent cerebral dysrhythmia and the beneficial, clinical effects of electroshock therapy. Correspondingly, studies were undertaken which involved a repetition of the electroshock experiments previously reported (6), but after preliminary injections of trypan red.

[3] In a previous study (4) it was pointed out that the term "blood-brain barrier" is confusing and that, if the name is to be retained, it should be restricted to the barrier between the blood and the extracellular fluid of the brain. The presence of another barrier beyond the cerebrovascular tree and probably located in the cell membranes of the cerebral tissue was stressed.

Methods

The studies involved determinations of the distribution of cocaine by spectrophotometric

TABLE 1.—Spectrochemical Determination of Cocaine Hydrochloride* in the Blood and Brain of Cats Pre-treated with Trypan Red,† Before and After a Series of Electrically Induced Convulsions

Treatment	Experiment No.	Cocaine Level (gamma/gm.)	
		Blood	Brain
Trypan red (no electric shock)	1	4.9	24.3
	2	7.9	28.5
	3	4.6	24.0
	4	—	20.4
	5	5.7	20.4
	6	8.6	28.6
	7	5.9	28.5
	8	5.1	25.0
	9	5.7	27.3
	10	6.9	32.3
Trypan red (after electric shock)	1	5.6	20.0
	2	4.9	21.3
	3	—	17.7
	4	5.1	20.6
	5	5.2	22.3
	6	5.4	27.0
	7	9.0	25.3
	8	6.5	21.6
	9	7.4	20.7
	10	5.1	27.3
	11	10.2	26.8
	12	4.8	27.0
	13	4.9	17.7

* Cocaine hydrochloride, 15 mg. per kg. of body weight, injected intravenously as described in the text.

† Trypan red, 1.65 cc. of a 1% solution per kg. of body weight, injected intraperitoneally. Four such injections were given over a period of from 5 to 7 days.

TABLE 2.—SUMMARY OF STATISTICAL ANALYSIS OF DATA ON DISTRIBUTION OF COCAINE HYDROCHLORIDE* IN THE BLOOD AND BRAIN OF CATS PRE-TREATED WITH TRYPAN RED,† BEFORE AND AFTER A SERIES OF ELECTRICALLY INDUCED CONVULSIONS

Specimen	Treatment	Number of Experiments	Mean Value of Cocaine (gamma/gm.)	Standard Deviation	Significance Test	Odds‡
Blood	Trypan red (no electric shock)	9	6.1	0.5	0.0	0:1
	Trypan red (after electric shock)	12	6.2	0.5		
Brain	Trypan red (no electric shock)	10	25.9	1.2	2.0	22:1
	Trypan red (after electric shock)	13	22.7	1.0		

* Cocaine hydrochloride, 15 mg. per kg. of body weight, injected intravenously as described in the text.

† Trypan red, 1.65 cc. of a 1% solution per kg. of body weight, injected intraperitoneally. Four such injections were given over a period of from 5 to 7 days.

‡ Odds against results being due to chance.

methods (1–5) in the brain and blood of cats receiving a series of electric shocks. In all respects the methods utilized in these experiments were identical with those used in the previous study (6) except for the preliminary injections of trypan red as indicated below.

A series of 12 electric shocks was given over a period of 22 days with the usual bitemporal placement of electrodes and with the use of an Offner electroshock apparatus delivering a current of 400 ma. for 0.2 second.

Electroencephalograms were obtained initially on all cats, after the seventh electrically induced convulsion, and again three days after the twelfth and final shock with the use of a Grass electroencephalographic apparatus.

Four intraperitoneal injections of 1.65 cc. of a one per cent solution of trypan red were given over a period of from 5 to 7 days, which approximated a dose of 10 mgm. per kilogram of body weight per injection. A fifth injection of trypan red (same dose) was given between the seventh and eighth electric shocks and 13 or 14 days prior to the final cocaine experiment.

Under moderate narcosis and in accordance with standardized techniques previously reported (5) the final cocaine distribution studies were conducted following the intravenous administration of 15 mgm. per kilogram of body weight of a six per cent solution of cocaine hydrochloride in both control and post-shock cats. In the case of the post-shock group, the cocaine studies were performed three days after the completion of the electroshock series of treatments. The cocaine was injected slowly and evenly over a 15-minute period. At five-minute intervals thereafter, specimens of cerebrospinal fluid, brain, and blood were obtained and immediately frozen in carbon dioxide snow. The brain and blood specimens were extracted chemically by standardized methods, which we have previously described (27), and finally analyzed for cocaine by means of a spectrophotometric technique, utilizing a Beckman photoelectric spectrophotometer.

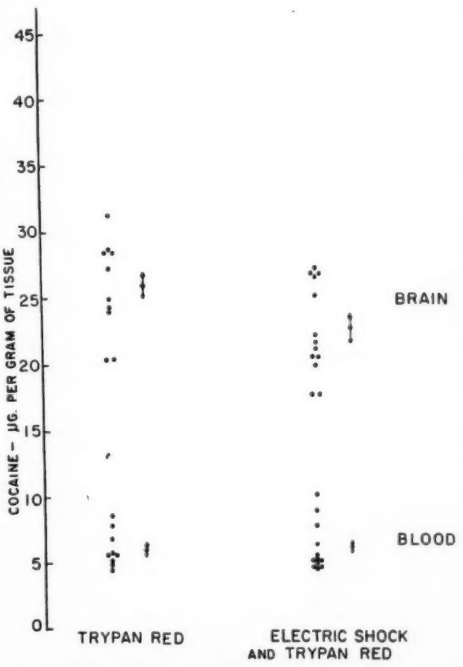

FIG. 1. Spot graph showing the spread of experimental determinations, the mean values, and the standard deviations of the means on the distribution of cocaine hydrochloride in the blood and brain of cats under control conditions and 3 days after a series of electrically induced convulsions.

Results

The results are presented in table 1 and are shown graphically in figure 1. An analysis of these data is presented in table 2.

TABLE 3.—EEG Findings in Electric Shocked Cats with and without Trypan Red Injections*

Experiment		Normal	Probable Normal	Probable Abnormal	Abnormal
Without trypan red	1st test (before shocks)	8	6	0	0
	2nd test (after 7 shocks)	1	8	4	1
	3rd test (after 12 shocks)	0	0	3	11
With trypan red*	1st test (before shocks)	8	5	1	0
	2nd test (after 7 shocks)	6	8	0	0
	3rd test (after 12 shocks)	0	8	1	5

* 1.65 cc. of a 1% solution of trypan red per kilogram of body weight was injected intraperitoneally. Four such injections were given over a period of 6 days prior to initial electric shock treatment, and a 5th injection was given following the 7th electric shock.

The findings indicate no alteration in the concentration of cocaine in the blood in the post-shock group of cats, as opposed to the control group. On the other hand, the concentrations of cocaine in the post-shock cortex (22.7 ± 1.0 gamma per gram of tissue) were slightly but not significantly lower than their corresponding control values (25.9 ± 1.2 gamma per gram of tissue).

The corresponding values for the cortex in the previous study (6), in which trypan red was not used, were 36.6 ± 1.4 gamma per gram of tissue in 14 post-shock experiments and 28.9 ± 0.9 gamma per gram of tissue for the control group (15 experiments). Thus, it would appear that trypan red effectively counteracted the effect of electrically induced convulsions upon the permeability of the blood-brain barrier. The post-shock values for cocaine in the cortex with and without trypan red were even more striking. The post-shock values with trypan red were 22.7 ± 1.0 gamma per gram of tissue in 13 experiments and without trypan red were 36.6 ± 1.4 gamma per gram of tissue in the 14 experiments mentioned above (6).

A review of the electroencephalographic

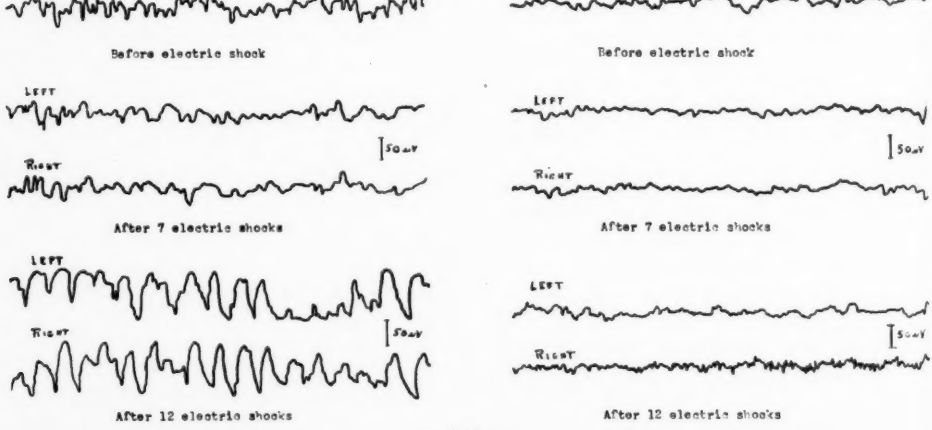

Fig. 2. Typical electroencephalograms of cats before and after electric shock treatments and with and without preliminary trypan red injections. Four intraperitoneal injections of 1.65 cc. of a 1% solution of trypan red given over a period of 6 days prior to initial electric shock treatments and a 5th injection given following the 7th shock.

results showed that the pattern observed after a series of electrically induced convulsions in cats, previously treated with trypan red, approximated the normal controls as opposed to the gross dysrhythmias found after shock in the previous study (6) in which trypan red was not used. In several of the trypan red experiments the pattern after electric shock appeared normal and essentially identical with the electroencephalograms obtained prior to the electrically induced convulsions. In other experiments a slight dysrhythmia appeared after a series of electrically induced convulsions, but in general, as may be seen in table 3, the electroencephalographic changes were relatively slight in the trypan red experiments. Representative electroencephalograms, before and after electrically induced convulsions and with and without preliminary trypan red injections, are shown in figure 2.

Discussion

The effect of the supravital dye, trypan red, in counteracting both the increased permeability of the blood-brain barrier and the cerebral dysrhythmia associated with electroshock is of great interest and suggests that the abnormal neurophysiology of electroshock, at least as evidenced by the generalized cerebral dysrhythmia usually associated with it, may be conditioned by the increased permeability of the blood-brain barrier. As mentioned previously, this same result was obtained in the studies on cerebral concussion (5). This result taken in conjunction with the findings of the previous study (6) would tend to corroborate the conclusions of earlier reports (1, 3, 5) that 1) the locus of the blood-brain barrier resides in the cerebrovascular tree, and 2) this basic neurophysiologic mechanism is capable of conditioning the neurophysiology of the central nervous system.

The failure of a cerebral dysrhythmia to develop following preliminary injections of trypan red, even though a series of electrically induced convulsions was administered which previously had been demonstrated as capable of producing such a dysrhythmia in another group of cats not treated with trypan red (6), is of particular interest.

For reasons already enumerated, this finding strongly suggests that the primary effect of the electric current was on the cerebrovascular tree rather than on the cerebral cellular masses supported by the vascular tree. This result in combination with the neuropathologic findings following slightly excessive electroshock currents, as previously mentioned, would make it appear likely that a high percentage of the current was transmitted along the cerebrovascular tree, or at least that portion of the current that produced residual neurophysiologic effects, which in a previous study (6) we have shown might be correlated with the somewhat persistent clinical effects of electroshock therapy. Since the finer cerebrovascular elements are distributed fairly evenly throughout the brain, this conclusion would not be contrary to the findings of the earlier experimental pathologic studies, which showed that the central core of the path of current transmission followed a fairly direct course between the stimulating electrodes, while the marginal spread of the current pathway showed an elliptical pattern.

In the light of these findings, suggesting a cerebrovascular transmission of electroshock current, it is of interest to review the results of other studies, which have been interpreted as favoring either a direct tissue (regardless of type or structure of the tissue) transmission of current, or transmission largely via neuronal tracts and especially the large intracerebral connecting tracts. These studies necessarily have depended upon average and gross determinations which do not permit a discrimination between conduction over neuronal as opposed to cerebrovascualr elements.

Although Alexander and Löwenbach (7) have interpreted their results as favoring a direct tissue transmission, they in part admit the influence of anatomical factors, including the cerebral blood vessels. In part, they have stated, "It is more likely that the lesser intensity of change in the median part of the central core of the path is also explained by anatomical factors. In the lateral parts of the core of the path, the current travels along the entire length of the larger supplying arterioles as they come in from the meningeal surface and distribute themselves within the medially adjacent gray and white matter, and the wedge-shaped outlines of the extensions of the areas of vasoparalytic stasis into the medially subjacent white matter are indeed more representative of the manner of distribution of these vessels than of the actual outlines of the core of the path of the current."

Direct measurements of intracerebral voltage have been made to determine the path of the current in electroshock therapy. These have shown that the high resistance of the skull and shunting of current through the extracranial tissues serve to diffuse widely the

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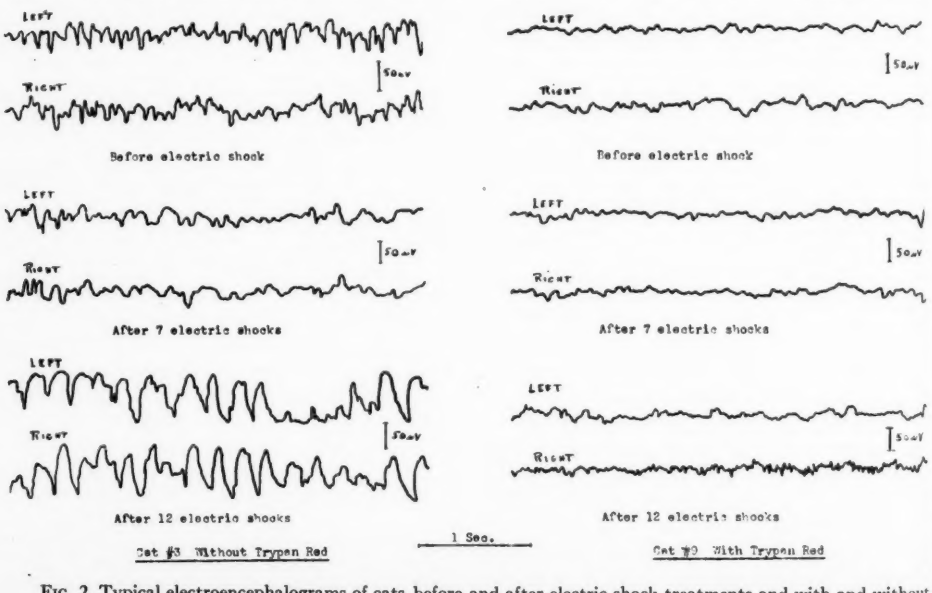

Fig. 2. Typical electroencephalograms of cats before and after electric shock treatments and with and without preliminary trypan red injections. Four intraperitoneal injections of 1.65 cc. of a 1% solution of trypan red given over a period of 6 days prior to initial electric shock treatments and a 5th injection given following the 7th shock.

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In the light of these findings, suggesting a cerebrovascular transmission of electroshock current, it is of interest to review the results of other studies, which have been interpreted as favoring either a direct tissue (regardless of type or structure of the tissue) transmission of current, or transmission largely via neuronal tracts and especially the large intracerebral connecting tracts. These studies necessarily have depended upon average and gross determinations which do not permit a discrimination between conduction over neuronal as opposed to cerebrovascualr elements.

Although Alexander and Löwenbach (7) have interpreted their results as favoring a direct tissue transmission, they in part admit the influence of anatomical factors, including the cerebral blood vessels. In part, they have stated, "It is more likely that the lesser intensity of change in the median part of the central core of the path is also explained by anatomical factors. In the lateral parts of the core of the path, the current travels along the entire length of the larger supplying arterioles as they come in from the meningeal surface and distribute themselves within the medially adjacent gray and white matter, and the wedge-shaped outlines of the extensions of the areas of vasoparalytic stasis into the medially subjacent white matter are indeed more representative of the manner of distribution of these vessels than of the actual outlines of the core of the path of the current."

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cerebral transmission of the small fraction of the applied shock which actually penetrates the brain. Smitt and Wegener (25) measured the potentials between probes inserted into the brain of cadavers subjected to electric shock, while Hayes (14) made similar determinations on an anesthetized monkey during electric shock. Unfortunately, the resistance between the probes was not determined with each measurement. Instead, the assumption was made that the resistance between the probes remained constant and that the potentials, therefore, could be related directly to current. The magnitude of the current following a given path depends on the conductivity (the inverse reciprocal of the resistance) of the tissue through which it is flowing, and this may well vary from area to area. Thus, potentials justifiably cannot be related directly to magnitude of current flow, and consequently the results obtained may be questioned. In any case, such gross measurements could not be expected to reveal whether the principal current flow is by way of vascular or neuronal pathways.

The study of Lorimer, Segal and Stein (20) suggests that the passage of current from side to side when the stimulating electrodes are placed "biparietally" is principally mediated via the deep thalamic structures, but affords little information as to the relative current densities in anterior-posterior dimensions, and again does not differentiate between possible vascular transmission as opposed to neuronal pathways. The rapid bioelectrical and biochemical changes that occur with death make it difficult to compare this study on cadavers with the findings of the present study.

The momentary stasis of cerebral circulation with electroshock therapy reported by Kreienberg and Ehrhardt (16) would in no way alter the validity of the vascular thesis of current transmission through the brain.

The vascular thesis of current transmission does not necessarily preclude some degree of transmission by way of the major intracerebral neuronal connecting pathways, such as the corpus callosum as suggested by the studies of Lorimer, Segal and Stein (20). Regardless of the primary pathway of transmission for the current of electroshock therapy, the stimulation of neuronal tracts and the spread of stimulation by neuronal pathways would presumably occur in the manner described by McCulloch (22) for the spread of epileptic activation, except as such neuronal pathways might be blocked if in a refractory state induced by the previous stimulation of the primary shock transmission.

The cortical and intracerebral electroencephalographic study of electroshock therapy by Delgado, Alexander, and Hamlin (11) suggests that the maximal transmission of the current occurs in a frontal plane corresponding to the bifrontal placement of the scalp electrodes used in the study. Although of interest in suggesting results similar to those obtained by pathological methods, the technique employed would not distinguish effectively between direct tissue transmission, vascular transmission, or indirect neuronal activation secondary to tissue stimulation.

More recent evidence that the hippocampus has a low electroconvulsive threshold (19) and that epileptic discharges may spread from the hippocampus to the pyriform cortex without involvement of the fornix or diencephalon (13) is of great interest and suggests that the neurophysiologic basis of electroshock therapy cannot be explained too simply in terms of any single mechanism. Differences in the convulsive threshold of different areas and the activation of the neuronal tracts associated with the regions of lowest convulsive threshold must be considered as well as the possibilities of vascular and direct tissue transmission of the current of electroshock therapy in explanation of the phenomenon. In addition, the relative vascularity of the hippocampus and the possible conditioning effect of altered vascular permeability (secondary to electroconvulsive phenomenon) on the neurophysiology of this region should be investigated further in view of the results obtained in this study.

Conclusions

1. Neurophysiological studies, utilizing a modified spectrochemical technique for following the distribution of cocaine used as a tracer agent, have been performed on cats previously treated with trypan red, both under control conditions and after a series of electrically induced convulsions. Cortical concentrations of cocaine were found in the cats subjected to a series of electrically induced convulsions which were equal to or lower than the corresponding control values. It was concluded that trypan red effectively counteracted the permeability effect of electrically induced convulsions on the blood-brain barrier.

2. A cerebral dysrhythmia failed to appear in the cats treated with trypan red in these experiments, or appeared only in minor degree

three days after the final electric shock. Since trypan red was found to counteract the increase in permeability of the blood-brain barrier as well as the cerebral dysrhythmia associated with a series of electrically induced convulsions, as also had been found to occur in another study concerned with cerebral concussion, it was concluded that the dysrhythmia and the abnormal neurophysiological state it reflects were conditioned by the increased permeability of the blood-brain barrier.

3. For reasons discussed, the failure of a cerebral dysrhythmia to develop following preliminary injections of trypan red, even though subjected to a series of electrically induced convulsions, which in a previous study had been shown to be capable of producing gross dysrhythmias in cats not administered trypan red, strongly suggests that the primary effect of the electroshock current was on the cerebrovascular tree, rather than on the cerebral cellular masses. This in combination with reported neuropathologic findings following excessive electroshock currents makes it appear likely that a high percentage of the current (or at least that portion producing residual neurophysiologic effects that might conceivably be correlated with the somewhat persistent clinical effects of electroshock therapy) was transmitted along the cerebrovascular tree.

4. The results of this study, when considered in conjunction with other studies discussed, indicate that the neurophysiologic basis of electroshock therapy cannot be explained simply in terms of any single mechanism. The results obtained, however, suggest that cerebrovascular permeability may be of importance to our understanding of the underlying neurophysiology of electroshock therapy, which conceivably might be of significance in explanation of the therapeutic value of this procedure. The possibilities include: (a) a vascular transcerebral transmission of electroshock current; (b) the importance of possible differences in the relative vascularity and in the convulsive thresholds of various areas of the brain; and (c) the conditioning effect of an altered vascular permeability (secondary to the electro-convulsive phenomenon) on the neurophysiology of regions important from a psychiatric standpoint.

BIBLIOGRAPHY

1. Aird, R. B.: Mode of action of brilliant vital red in epilepsy. *Arch. Neurol. & Psychiat., 42:* 700–723, 1939.
2. Aird, R. B.: The effect of desoxycorticosterone in epilepsy. *J. Nerv. & Ment. Dis., 99:* 501–510, 1944.
3. Aird, R. B., and Strait, L.: Protective barriers of the central nervous system; an experimental study with trypan red. *Arch. Neurol. & Psychiat., 51:* 54–66, 1944; and correction of graph, *Ibid., 53:* 67, 1945.
4. Aird, R. B., and Strait, L.: Mode of action of sodium diphenylhydantoinate (dilantin) in epilepsy. *J. Pharm. & Exper. Therap., 103:* 136–146, 1951.
5. Aird, R. B., Strait, L. A., Zealear, D., and Hrenoff, M.: Neurophysiological studies on cerebral concussion. *J. Neurosurg., 9:* 331–347, 1952.
6. Aird, R. B., Strait, L. A., Pace, J. W., Hrenoff, M. K., and Bowditch, S. C.: Neurophysiologic effects of electrically induced convulsions. *Arch. Neurol. & Psychiat., 75:* 371–378, 1956.
7. Alexander, L., and Löwenbach, H.: Experimental studies on electroshock treatment; the intracerebral vascular reaction as an indicator of the path of the current and threshold of early changes within the brain tissue. *J. Neuropath. & Exper. Neurol., 3:* 139–171, 1944.
8. Alpers, B. J., and Hughes, J.: The brain changes in electrically induced convulsions in the human. *J. Neuropath. & Exper. Neurol., 1: 173–180,* 1942.
9. Cerletti, U., and Bini, L.: Le alterazioni istopathologiche del systema nervoso in seguito all'E. S. *Riv. sper. di Freniat., 64:* 311–359, 1940.
10. Corsellis, J. A. N., and Meyer, A.: Histological changes in the brain after uncomplicated electroconvulsant treatment. *J. Ment. Sc., 100:* 375–383, 1954.
11. Delgado, J. M. R., Alexander, L., and Hamlin, H.: Effects of electroshock on the cortical and intracerebral electro-activity of the brain in schizophrenic patients. *Confinia neurol., 13:* 287–294, 1953.
12. Gralnick, A.: Fatalities associated with electric shock treatment of psychoses; report of two cases, with autopsy observations in one of them. *Arch. Neurol. & Psychiat., 51:* 397–402, 1944.
13. Green, J. D.: Discussion of Electroshock and rhinencephalic seizure states, by W. T. Liberson and J. G. Cadilhac. *Confinia neurol., 13:* 284–285, 1953.
14. Hayes, K. J.: The current path in electric convulsion shock. *Arch. Neurol. & Psychiat., 63:* 102–109, 1950.
15. Hyslop, G. H.: Distant secondary circulatory and vasomotor reactions to accidental electric shock. *Bull. N. Y. Acad. Med., 21:* 302–306, 1945.
16. Kreienberg, W., and Ehrhardt, H.: Die Gehirndurchblutung im elektroshock. *Nervenarzt., 18:* 154–163, 1947.
17. Langworthy, O. R.: Nerve cell injury in cases of human electrocution. *J. A. M. A., 95:* 107–108, 1930.

18. Langworthy, O. R.: Abnormalities produced in the central nervous system by electrical injuries. *J. Exper. Med., 51:* 943–964, 1930.
19. Liberson, W. T., and Cadilhac, J. G.: Electroshock and rhinencephalic seizure states. *Confinia neurol., 13:* 278–286, 1953.
20. Lorimer, F. M., Segal, M. M., and Stein, S. N.: Path of current distribution in brain during electroconvulsive therapy; preliminary report *EEG & Clin. Neurophysiol., 1:* 343–348, 1949.
21. MacMahon, H. E.: Electric shock. *Am. J. Path., 5:* 333–348, 1929.
22. McCulloch, W. S.: Mechanism for the spread of epileptic activation of the brain. *EEG & Clin. Neurophysiol., 1:* 1924, 1949.
23. Morrison, L. R., Weeks, A., and Cobb, S.: Histopathology of different types of electric shock on mammalian brains. *J. Indust. Hyg., 12:* 324–337, 1930.
24. Neubuerger, K. T., Whitehead, R. W., Rutledge, E. K., and Ebaugh, F. G.: Pathologic changes in the brains of dogs given repeated electrical shock. *Am. J. Med. Sc., 204:* 381–387, 1942.
25. Smitt, J. W., and Wegener, C. F.: On electric convulsive therapy with particular regard to a parietal application of electrodes, controlled by intracerebral voltage measurements. *Acta Psychiat. et Neurol., 19–20:* 529–549, 1944–45.
26. Spatz, H.: Die Bedeutung der vitalen Färbung für die Lehre vom Stoffaustausch zwischen dem Zentral nervensystem und dem übrigen Körper. *Arch. f. Psychiat., 101:* 267–358, 1934.
27. Strait, L. A., Aird, R. B., and Weiss, S.: A method for the rapid isolation and spectrographic measurement of cocaine from brain tissue. *J. Pharm. & Exper. Therap., 73:* 363–374, 1941.

MENTAL DANGER, STRESS AND FEAR
A Semantic and Clinical Inquiry

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In the etymological history of a word we find manifold shades, meanings and genesis of concepts. When we hear or read those tokens of expression called "words," we unconsciously associate those hidden meanings with them. Such exercise may help us toward a clearer description of feelings and concepts we want to explore. The knowledge hidden in words predated psychological or chemical understanding of danger and fear. By linking the purely descriptive semantic data with clinical dynamic description, I hope at the same time to integrate the phenomenological approach with psychodynamic concepts.

A great difficulty that arises in an attempt to describe man's various reactions to danger[1] lies already in trying to define what danger is. Small dangers help to form man's personality; big dangers overwhelm and destroy him. Although we know what various physical dangers —such as too much heat or cold, poisoning, invasion by bacteria and viruses—do to the physical organism, it is more difficult to define what agents injure the mind. Although the concept of danger is related to that of fear and anxiety, it needs its own investigation and may give new insights in both concepts.

Our word danger is, interestingly enough, derived from the danger of mental pressure. It comes from the old French *dongier* and this from the Latin *dominarium*, the peril of being subjected to domination and jurisdiction by authority. The German *Gefahr* is more directly related to fara, that which arouses fear. Old German *Faer* also means the sudden peril of travelling, of separation. Peril comes from the Latin *periculum*, meaning a trying out, testing, experimenting with something unknown, while the word hazard comes from the Spanish *azar* or *asart*, a game of chance.

Etymologically we find the concept of mental pressure and the gamble with the unknown expressed as conditions of danger. We must point out, too, that originally no sharp distinction was made between what constituted a danger to the mind and what to the body. The growing science of psychosomatic medicine has shown very clearly that there is a mutual and inseparable relationship between mind and body. There are mental influences damaging to both; there are physical injuries affecting both. If you burn your finger, you react to the danger by suddenly withdrawing your hand from the fire. If the burn is severe, the skin will react by forming blisters. But, in the meantime, mental processes take place which warn you against repeating this experience. In a very sensitive person, a real fear—of stoves or gas burners, for instance—may develop as a result of one such experience, though every animal knows instinctively what such physical danger consists of. In general we may say that after one noxious experience the organism is prealarmed, and there may be increased irritability as a warning against a repetition of the upsetting experience. There are various individual differences, dependent on a greater or lesser alertness to danger.

Fantasies and distorted anticipation of danger may have an even greater influence on the reaction of the organism than the actual impact of danger. An example may illustrate this. During the Second World War, I was playing tennis with some friends. It was during the German occupation of Holland, and on a tennis court next to ours German officers were playing. Suddenly a squadron of British Spitfires came over, flying rather low. My friends and I cheered them—because they were the allies we hoped would liberate us—but the German officers threw themselves on the ground in their white tennis outfits as though in sudden panic. Had the British bombed or strafed us, both the Germans and my party would have been exposed to the same danger, but subjectively the situation was different. For the Germans the fliers were the enemy; for us the friends. Half a year later—after I had escaped to England—when I experienced

[1] A systematic description of man's various reactions to danger will be presented in a subsequent study.

German bombardment of London, I had the same slight paranoiac feeling as the German officers had had on the other side of the English Channel, that every bomb was meant for me.

During such bombardments we experienced only few direct panic reactions. However, those people who heard about the bombardments by radio or newspaper, because they lived away from the direct calamity, constructed in their fantasy danger images with far greater implications for their mental equilibrium. In them, the sensation-laden bystanders, we saw examples of real hysteria.

People who went through the actual calamities of war often developed pathological danger reactions (i.e., fear reactions) much later. They went courageously through hell, but later on, when fantasy and unconscious feeling could quietly partake of the process, the courageous adjustment to a threatening world could not be sustained. They let themselves go in desperation. Fantasy about danger often produced more disturbing reaction than the reality of it.

The Law of Life's Total Reaction

The rather simple example of alertness toward danger shows that danger evokes reactions in several parts of the organism and not only at the point of direct impact. We owe our best knowledge of these facts to the investigations of Goldstein (6) and Cannon (2). Even widely separated parts of the body may contribute to the general defense of the organism. Thus, we experience physical reactions to mental stimuli and *vice versa*. During recent years much has been written about the conversion of mental reactions into bodily expressions, and about the psychological changes that take place in the wake of physical injury. As a result of our new understanding of this "total" psychosomatic reaction, the purely somatic and mechanical point of view that used to dominate the science of life in the beginning of the century has all but vanished from medical thinking.

Every living being is a reactor in continual exchange with the outside world. Life has essential qualities still inexplicable in purely physical or chemical terms. Scientists are fascinated by these different facets of life. Some emphasize the individual quality of every living thing—there are never two completely identical living individuals—but in dead matter the law of equality and uniformity rules. Others are especially interested in specific life processes such as respiration and procreation—no engine ever procreated itself or defended itself actively against danger. Some see this defensive activity against intrusion by outside forces as the quintessence of life. When these forces are too strong, and break through the established defenses, we may call their intrusion: danger.

Again, another scientist may concentrate his work on the continual changes that take place in human beings during the process of adaptation and adjustment. Dead matter is affected only by the laws of chemical decomposition and decay. But life has a tendency to conservation, self-preservation, and stabilization.

Still other scholars may point at the particular quality of the life process, which counters the mechanical processes with a special "life strategy".

Life is something with a unique biological history, a process that is not simply reversible like chemical reactions. Here again we meet the concept of danger in the failure of the "life strategy" and its resulting transfer into purely physico-chemical processes—death. Where there is life, there is danger, because of the possible intrusion of forces destroying the unique structure of life. This corresponds with the concept that ultimate danger means the loss of structural quality of life, of potential energy, resulting in death. Loss of structure means the surrender to the realm of chaos and random probabilities.

Other students see the autonomy, the self-regulation of life as its defining characteristic in which the principle of trial and error operates. But every living organism has its own limitation and endangers itself when exceeding it. We may reformulate it in this way: Life has a self-assertive, growing quality expressed in the processes of incorporation (eating) and multiplication and reproduction (sex). Frustration of these biological processes may be called a danger to life. Protoplasmatic hunger (aggression) and reproductive tension (libidinal urge) are the fundamental activities of the cell. Here already begins an archaic biological periculum; danger starts with birth, and from birth-trauma to death-fantasy man plays continually with the concept of danger and survival.

However, these simple examples of life as a living reactor have, in the case of human beings, to be expressed in more complicated and subtle terms. Man is a vertebrate, a mammal, a primate; yet in many ways he cannot be compared with lower animals. In addition to the normal biological limitations, man has typical human limitations and prerogatives. He is the only animal that uses thinking and verbal skill as preparation for action. He has a practical intelligence, makes his own tools, and has a sense of distance and humor, of memory and judgment.

"Man is, on the whole but not in every single respect, the pinnacle so far of evolutionary progress" (21). That is to say, in this respect he is the most adaptable being and the most progressively independent of the environment. In him "a new sort of evolution has appeared, a new sort of heredity, the inheritance of learning." "In the new evolution we can inherit directly from ancestors of two thousand years ago, or from ancestors of more recent date, perhaps, who lived half way around the world from us or from our immediate kin," according to Simpson. "Human social organization is in a sense the basis of the new evolution . . . "—an evolution brought under conscious control. Man has an innate instinct for curiosity, and innate social instincts. He wants to belong somewhere, to play a role in his society; he looks for mutual exchange, and anticipates interrelationships.

The greatest danger for man is the failure of his social organization, the failure of his typical human adaptabilities, as we see, for instance, in war, in social frustration, and discrimination. Yet, gradually, man's disciplined mental activity increases his adaptability. Psychology has been able to express some new dangers belonging specifically to man. Distinguishing himself from other animals by a longer dependency on his parents and a longer youth, he received, in exchange, a nearly unlimited learning capacity. However, man's *dependency necessity* provoked new dangers; the danger of too early a separation from and deprivation of the protected home; lack of food and warmth; lack of affection and security which may cause the danger of too early sexual rivalry with one of the parents. Incest-taboo and oedipal complex protect the vulnerable human youth against those typical human dangers.

Clinical experience in neuroses can provide many more examples of such typical human dangers, such as sibling rivalry, too early weaning, automatic conditioning, lack of love, and so on.

In contrast to the instinctual innate knowledge of animals, human knowledge is cumulative and progressive, thanks to man's ability to communicate his experiences. Man is aware of a history of his life. Knowledge of forces can teach him to use these forces or to eliminate them. Man's *living knowledge* makes him able to seek for newer and better patterns of adjustment. As Korzybski (10) expressed it, human life is a time builder, man is able to accumulate the experiences from mankind's past in his own individual mind. Man, a product of nature, learned not only to understand and, in many ways, to master nature but also to acquire an idea and comprehension of himself. He needs such comprehension in his struggle for life: lack of it can become a new danger to him.

Man learns not only from his own experience; he is able to use other people's corrections and experiences. Limited learning may become a danger to him as we experience so often in social isolation. Yet school and education, all that takes man away from direct instinctual satisfaction, are often regarded as danger. A tremendous fear of intellect and acculturation exists. In order to learn, man must live in a social world. His experiences are transmitted. The global process of mutual interdependence may be interpreted as a collective aim towards avoidance and conquest of danger. Beyond this, man lives in an imaginative world; he tries to prepare for the future, he knows introspection, he gains and ever-growing consciousness of himself, he communicates with his fellow beings—he is a creative animal.

All these human qualities influence his reactions to danger and may help him to surpass his animal limitations. Being human demands not merely defense but also higher forms of social adaptation and adjustment; these imply new forms of danger.

Experimental Danger

In an experimental situation, external and internal dangers (outer conflict and inner anxiety) can be suggested. I myself was able to carry on such experiments by suggesting anxiety-provoking dreams or by submitting

students to various toxics. This goal could also be achieved by arousing post-hypnotic conflicts. During the war, anxiety-provoking suggestions were used to work out soldiers' deeply-rooted battle fears. As a tentative result we found that various small groups reacted in completely different ways to the anxiety-provoking situation, often depending on the reaction of a dominating neurotic personality in the group. Such a personality would express his private fear and anxiety and contaminate others before they could make up their own minds.

Individuals in the experimental situation reacted to such situations according to their own past conditioning. How varied the reactions are in such groups and individuals is a surprise to the novice experimenter. In the simplest experimental situation—e.g., suggesting a hospital move nearer to the battle line—different reactions come to the fore. One person reacts with generalized fear, trembling and exhibiting the need to escape; another with organ reaction like vomiting, stomach ache or diarrhea; a third with denial and blind-spots. In the latter, evading fantasies and negative hallucinations predominate; for him there is no danger. Another one intellectualizes the situation and assumes a pseudo-philosophical attitude, replacing danger by verbosity. Still another may react with direct attack and storm the bastion —"Come let us go!"

However, the unsteadiness of the patterns in those wartime experiments impressed us. The same person, during another group session, would react differently. *The same dangers coming at different times in our lives have different meaning and impact for us.*

I want especially to emphasize here that future experimental work with groups and individuals about experimental stresses will give clearer insight into basic reaction patterns to danger and the varied strategy of body and mind.

Stress

In the search for a better understanding of what comprises the concept of danger, scientists could use some of the comparable concepts from the science of physics. In physics one calls *stress* the total amount of physical forces acting on a body—e.g., the weight resting on the pillars of a bridge. The reaction caused by the stress is called *strain*. Within limits, called the elastic limit, the strain will be proportionate to the stress. Man also has a comparable elastic limit beyond which totally different processes occur. We experienced that in the last war. There are very different limits of endurance. Some people broke down under the slightest impact of danger; others hardly ever broke down, or if they did, in the midst of the holocaust, they recuperated very soon.

Yet, in biology the simple relationship between stress and strain, as found in physics, has changed into a far more complicated one. In the first place we cannot speak of simple stresses acting on the body. Sometimes they may be purely physical, chemical, bacterial or metabolical, but beyond these we have to take into account conditioned stresses as a result of psychological training, learning, and specific life experiences. Special stimuli, as for instance hunger, can become triggers for old, conditioned reactions to such stresses as could be experienced in famine reactions. Biologic behavior is partly a symbolic behavior, there is present not only stress but also an image of stress, a notion and an anticipation of stress, often resulting in the same kind of strain.

This complicated addition of symbolic and conditioned stresses to the general concept of physical stress I want to call "danger".

It must be said that the response of the organism itself is not simple strain either, but a typical biological change of equilibrium resulting in adjustive behavior, such as food-seeking behavior or avoidance reactions. However, the result may also be a breakdown of adjustive behavior as we experience in the so-called shock-like reactions.

In men, many of the stabilizing reactions are, as we have already seen, under control of the ego, the hypothetical central structural force that among other functions preserves the constancy (equilibrium, homoeostasis) of internal conditions. Within recent years we have learned to understand more about these various avoidance-approach reactions and their mutual influences.

In man the source of danger is for the greater part displaced from external stress to stress caused by internal systems of adaptation, inner stability and automatization of functions. From those internal changes, no withdrawal is possible. Every adaptation, when becoming too rigid, may turn into a liability when faced

with a new stress. In cases of such rigid adaptation special problems of expenditure of energy come to the fore as we have learned from the study of feedback mechanisms. The more compulsive exactitude (obedience) is asked for, in order to reach a goal, the more energy is required (much more, proportionately). This is not a simple arithmetical ratio. The exactitude of measure is proportional to the quadrupling of the original energy. In order to get twice the exactitude, one has to pay four times the price in energy. This may give us insight into how much energy is spent in compulsive adaptation, and compulsive control may become a serious danger for the body. (In my book, "The Two Faces of Man" (17), I investigate these problems of compulsive adaptation in a more extensive way.)

extreme temperatures cause damage to the psychosomatic organism, so certain psychological occurrences may be injurious. Among the principal injurious emotions are those aroused by the experience of birth, departure, isolation, separation, fear, sorrow, and catastrophe. Especially, the danger of separation and not belonging is experienced as tremendously traumatic. The family, the group, the nation have acquired a protective function in human society as a substitute womb. Excommunication is like dying. In this primary sense, submission to collective rule becomes a protective device. We may look at masochism as rooted in such primary submission to avoid greater danger.

From war experiences we learned that the awareness of lack of inner defensive power and

Resumé

The concept of danger implies:	*Strain* Tolerance-Capacity	Weak instinct of self-preservation
		Physical strain: exhaustion / internal expenditure of too much energy
		Homeostatic change: shock / sickness
	External Stresses Objective Danger	Environmental forces
		Deprivation, separation
		Noxious stimuli
		Abstention and sudden change
		Annihilation threat
		Accumulation of little stresses (the pin pricks)
	Internal Stresses Internal Danger	Weakness (homeostatic lack)
		Compulsion
		Sickness
		Destruction of the ego
		Overwhelming by instinctual impulses
		No mental backbone
	Symbolic Stresses Subjective Danger	Symbolic triggers (unspecific)
		Thinking
		Anticipation, Rumination
		Loss of love (separation)
		Castration. No phyletic continuity
		Internalized dangers
		Guilt and shame (alienation of superego)
	Conditioned Stresses Fantasy Danger	Lack of education and initiative
		Between mastery and delayed response a controlling ego develops
		Training to deny danger
		Cultural ambiguity
		battle-neurosis—avoidance of danger and cowardice at the same time
		Repression of emotional discharge

What Is a Psychological Danger?

First of all, every physical danger is also a psychological danger, often made more severe by inner distortion through fantasy and anticipation. However, just as bacterial toxins or lack of forces to identify with means the greatest danger to us. Many victims, taken by the Nazis from their homes and put into prison or camp, experienced this as an awareness of nothingness, a lack of backbone or steering

ego. As a matter of fact, for days they felt as if they had no human structure any more (depersonalization syndrome) because the endangering powers were too overwhelming. There was no immediate feeling of fear, only of nothingness.

There are many mental poisons that belong to this category of psychological dangers: hatred, envy, boredom, lust for power.

Is it possible at this time to point out some of the typical human dangers? We can give a tentative survey—as I will try to do in this paper—although we will find most of our answers in a better way after we have conducted a more scientific study of the various danger reactions to which man is prone.

The Interplay between External and Internal Dangers

On the basis of his clinical experiences, Freud (4, 5) directed our attention to the peculiar interplay between the feelings of external and internal danger. In man, the subjective dangers from within the depth of the person—from frustration, old feelings of guilt, archaic mythical horror fantasies—often make people much more vulnerable than the objective dangers threatening from outside. Indeed, in the last war we learned that the loss of love and affection and the breaking of family ties affected children more than the outer destructive aspects of war. The evacuation of their homes, leaving their parents, disruption of their accustomed daily life—these things upset the children but not the bombing and the mechanics of destruction.

In neurotics we experience particularly the role of the internalized horrible fantasies; originating in childhood, these hidden fantasies only come to the fore in dreams and nightmares.

Objective, recognizable dangers, such as floods or even bombing, stimulate the psyche to become alert and to set up defenses. But imaginary or unseen dangers—such as caused by rumors—overshoot the mark; they imply greater inner uncertainty and stimulate the formation of neurotic defenses and fearful anticipations.

They cause man to be continually mobilized and on the alert and so make him more vulnerable. Such inner fears are often related to the fear of one's own repressed instinctual drives and to hidden feelings of hate, guilt and aggression. We so often meet neurotics who cannot face their tremendous hostility and who convert it into continual panic and anxiety. In brainwashing, hidden guilt of this kind is used by the enemy to break down the inner strength of his victims. By threatening the victim's family, his friends and his buddies, he appeals to that tremendous reservoir of unconscious feelings of guilt in man.

In our civilization a tense equilibrium is maintained between internal and external dangers. "Stop" and "Go" signs become confused since so much is artificial and so many diversified, confusing choices are left to the individual. Sometimes the dammed-up aggressive forces may burst out beyond all control even when there is no real danger. We experience this in the many forms of unexpected rage and fury inspired by fear. Sometimes the danger from outside may be so very great that it tears through all our cultural defenses, and we break down. The primitive and helpless child in us takes over.

However, psychiatric experience leads us to believe that what breaks man down is, for the most part, the already existing lack of inner stability and control, although external disaster may provoke and touch off such internal disturbances as we see in battle neuroses.

As an example of this, during the last war, an extremely courageous fighter pilot asked for psychotherapeutic treatment because he found himself trembling and overcome by fear. He had flown more than forty dangerous missions without feeling at all afraid. However, during his furlough to town he had been forced to hide in a shelter because there was an air alert. It was this unforeseen sojourn with panicky people in a small bomb shelter with their contagious fear that had completely broken him. In the course of discussions with him, the fact emerged that various additional personal conflicts had brought him to his point of sudden breakdown. A conflict with his commanding officer, a sexual conflict with his girl friend the previous night, having been thrown together with other persons who were anxious and trembling in a small dark shelter—all these coincidental occurrences had aroused deep, hidden fears which were apparently only slightly related to the real, acute danger of the moment.

Do not think that this is only a negative finding. Inner strength is somewhere related to the imprinted strength and experienced happiness during the early years at home. The internalized childhood experiences determine our actual capacity for endurance. Those who have happy memories to build on are stronger in life than those who reminisce childhood unhappiness. Being happy is an early conditioned inner art; it cannot easily be provoked from outside. Inner strength is largely determined by the number of happy memories we have to live on. These again are dependent on the parents, teachers and leaders we can identify with and the training in discipline, consistency and endurance they provided for us.

It is important to realize that various inner factors may cause people to use external dangers as justification for inner confusion and breakdown. Our pilot walked around with an inner conflict for a long time. In his active work as a fighter-pilot he could get rid of his rage and resentment. But being passively delivered to the anxiety of others was the last straw—that broke the camel's back.

The weakling in us may use every outside danger to become a dependent, self-pitying child. Now and then everybody runs into a situation in which he finds that outside catastrophe only acts as a trigger or spark setting off hidden tensions and emotions so that they rise to the surface.

The Reminiscence of Archaic Dangers

Primitive man lived in constant danger and mental alertness; he lived in a world of continual fear, softened by the imagined protective influence of his gods. Not only were wild animals and the uncontrollable elements his enemies, his neighbor, who could slaughter and eat him, was too. "Eat or be eaten" is the old law of the jungle. It signified the tremendous danger of death and annihilation. Part of our fears is determined by the memorization of phylogenetic, ontogenetic and collective injuries: they are the big warning signals of pain and destruction.

In times of war and catastrophe it is as if those old latent fears of primitive man and the helpless infant revive in us. Our past is not forgotten. And there are many circumstances, in wartime for instance, that provoke such long-buried feelings of the race. What are those elementary dangerous experiences that give rise to overwhelming fear?

1. *The danger of being born.* The trauma of being exposed to new dangers and new circumstances in general, with its implications of the urge to regress to a state of rest and equanimity.

2. *The danger of suffocation.* This is the great reminiscence of the trauma of birth. The threat of gas warfare reminds us of this first threat and the fear of death. Now even the air we breathe is no longer safe. That is why gas drills have been known to induce panics in the participants.

3. *The danger of falling.* The fear of being like a helpless, not yet coordinated baby, which is an expression of utter dependency needs. It is also represented by the fear of being swallowed up, of being lost, uncoordinated, of not belonging anywhere.

4. *The danger of unexpected noises.* The crashing of bombs, heavy explosions, gunfire, or the sound of air-raid sirens have a violent effect on the ear and, relayed to the labyrinth, will induce giddiness or even complete unconsciousness. Such noises may have represented the damning voices of the primitive ancestral gods or the punishment of the Father of Thunder and Jupiter Tonans. They were the first sounds to startle the child. The French attribute the "commotion emotionelle" at the battlefront, which resembles cerebral concussion, to the physical and psychological influence of the continual intense noise of guns and explosions. Man's sense of hearing is extremely delicate, and constant jarring weakens its power of resistance. This can be seen in soldiers who have been exposed to lengthy bombardment or heavy barrage. Once outside the range of the noises, they may recuperate very soon.

Points three and four are also related to the sensitivity of the vestibular apparatus as a defense against danger. Dizziness represents loss of equilibrium, being overwhelmed and annihilated by space. Every threat to man's actual status, to his career and social integration can make him dizzy again.

5. *The fear of mysterious, hidden forces. The fear of the world's decline, the fear of earthquakes.* Now Mother Earth has become the dangerous enemy. The same concept lies behind the fear aroused by the atom bomb. The indiscriminate

total destruction of modern warfare has much the same effect on us as an earthquake. It makes people feel that there is no safety to be found anywhere, above or below ground. In bomb shelters, locked up in a small place with a number of other frightened people, fear is often increased. In the same way occurrences in the sky, such as meteors and comets, can cause enormous fear in those who do not understand what those phenomena actually mean. In Hiroshima, the tremendous flash of the atomic bomb aroused more fear than did the actual destruction it caused. For a long time the people of that city were hypersensitive to every sudden light-flash, and each time they saw one, they sought shelter again. Man's awareness of mortality, the certainty of his future death are related to these fears of Nemesis.

6. *Other hereditary dangers.* The image of danger goes through the whole history of mankind. Catastrophes live on in myths and saga, in many old superstitions. They are communicated from one generation to the next and live on in history. Are they inherited, or are they simply communicated by the cultural setting? It is not our task to solve that problem. Among these fears are: the fear of spirits, especially the spirits of the dead (of the slain ancestors); fear of aggression, of starvation, of famine and cold. Some psychologists may explain this as a phylogenetic memory, as hereditary remnants of the catastrophe of the ice age. Our world is still full of these dangers: the fear of water and floods, of wild animals, the spirits of totem animals, the feared "old men", ghosts and witches, devils and gods.

7. *The danger of deprivation and separation.* Mobilization and war separate men from their loved ones. In infancy, separation and rejection are the greatest dangers of the biologically dependent child. The first step and every new "step" in life is a new separation. Besides these physiological separations, real loss of the loved ones takes place; the dead ones may haunt the survivors; guilt and lack of libidinal satisfaction develop. The psychology of old age is fraught with this idea of final separation.

8. *Revival of cultural fears.* Fear of the wish for regression, fear of guilt and frustration. Fear of the so-called bad internal objects, of the sudden outburst of repressed instinctual hate and destructivity. The fear of being misjudged and not accepted.

The Traumatic Experience and the Trigger Action

Small causes may have tremendous effects. A minor experience may be the beginning of a completely changed life. In many nervous disturbances we find only a small direct "cause", the traumatic experience that precipitates hidden neurotic patterns of adjustment. It is as if the psychosomatic organism had waited for the small emotional trigger to start an inner avalanche. The traumatic occurrence has, as it were, a retro-causal action by giving new emphasis to past experiences. Today changes the past as much as the past determines today.

Only in recent years has scientific medicine been able to prove that there is no essential difference between physical and mental danger. The mere awareness of danger, of being emotionally upset, may in itself at once mobilize our inner defenses and, for example, increase the adrenocortical hormones in the blood (19). Through this adaptation mechanism mental danger already has its physical equivalent.

Selye has tried to generalize the concept of mental and physical reaction to danger or stress. He calls the answer of the organism to an outside stress the *general adaptation syndrome*, in which he shows three different stages.

The first one may be called the *alarm reaction*. On the basis of sudden preventive measures the body goes into a state of shock. The adrenal and pituitary glands are in a phase of overwork. The organism prepares, as it were, to discharge the invading stimuli.

The second stage is the *resistance reaction* in which the organism builds up increased resistance often at the cost of defense to other dangers. It resists but may become more vulnerable at the same time. The invading noxious stimuli cannot be discharged completely, they have to be tolerated, incorporated, and may be voided later on. A traumatic mnemic impression is formed.

The final stage may be *exhaustion* in which the resistant factors break down and the state of shock and alarm returns.

Pathogenic Communication and Mental Infection

Since symbolic dangers are brought to us by means of communication, communication plays a tremendous role in actual fear and panic.

Life is full of small causes which may provoke serious disturbances in the mind. We often call them pathogenic suggestions or communications. Tongue and language have partly taken over the old aggressive means of the animal. I remember a man who was rejected by his sweetheart; herself far from a dainty girl, she had always sneered at him because of his ugly face. There was no real basis for the sneering; she did it only to get rid of him. But after the separation he could not free himself from the verdict that he was an ugly misfit. Of course, his hurt pride and some of his earlier childhood experiences played a role in his neurosis. But for him, the girl's acrimonious suggestion had been the dangerous trigger.

These pathogenic communications and suggestions play an even more important role in social formations, where, often, the unusual or pathological behavior of one member may infect the others with abnormal ideas. This phenomenon can be seen very easily in isolated communities. In one case I know of, this kind of situation in a small town resulted in religious murder and suicide. In order to find more examples of the tremendous impact of communication we do not have to go back to the many accusations of witchcraft in the past. Isolated, lonely communities are as full of mental pathology as sailors may be on far flung expeditions or lonely planters in the tropics. Cold war and psychological warfare have shown us even more clearly how contagious pathological thoughts can be.

A wrong interpretation of a man's feelings and sinful thoughts, or a false accusation may bring a conscientious person into a depression, and may sometimes lead to suicide. Instead of giving psychological understanding and encouragement, the world often answers with hypocrisy and pitiless moralizing. I once knew a boy who went so far as to attempt suicide because of his struggle with masturbation and his anticipatory fear of his parents' attitude toward him. As his fear increased, his masturbation increased in reaction, and so did his self-punishment and self-rejection.

The phenomenon of suggestion and transfer of feelings by means of communication gives us more insight into the nature of mental infection. A religious or political maniac, possessed by his delusions, can infect large masses of people with his madness. He can incite them to fear, anger and abnormal actions. During a period of such continual mental contagion, every member of the infected social formation is temporarily changed. Fear and panic are especially contagious emotions. As long as the dangerous suggestion works, most men are unable to criticize and check their own actions. Modern psychology is becoming more and more aware of the fact that parents transfer their fears and insecurities to their children without being aware of their baleful and noxious influence.

The process of mental contagion from pathogenic sources occurs almost unconsciously. But we have to keep in mind that it is part of the process of mutual exchange and mutual influence which goes on constantly among human beings. Emotions influence us, sudden movements of other people, yawning, the rhythmic passing of soldiers, the boredom or excitement of a conversation, the rhythm of music, the gestures of our company, the voice of the radio, the sly and insidious fascination of television. Especially, an inner sensitive attitude makes people over-emotional in their relations to others. Inner guilt or fear of attack impels them to project on their opponents qualities they do not have. Slight gestures are interpreted as disagreements; even words of admiration may be interpreted as mockery and criticism. Such suspicion may be very contagious. It is this particular form of human contact that may become a danger in itself. We continually take over the habit patterns of our fellow beings. Thus, our words and ideas can become infected. Cold war and propaganda turn truth into poisonous catchwords. The Second World War taught us that the fear the parents had instilled in their children had a far greater influence upon them than the horrible reality of destruction and objective danger.

Mental Terror

By making use of terror, every dictator takes advantage of the contagious action of the means of communication. Goebbels' ministry of

propaganda taught us how even the selective editing of factual war news can stimulate and deceive an entire population. And, when this was not sufficient, mental terror finished the job and made the masses easy victims of the delusions of the dictator.

Terror is a serious psychological danger, causing a lie, repeated ten times, to become an indelible, stamped-in truth. The human intellect is unstable and prone to surrender to power. Our everyday resistance against crazy suggestions is rather weak.

Terror makes it impossible for us to control our thoughts through our intellect and our conscience. First, people become silent and over-tolerant. Fear and fright condition them to suffering. Gradually, they repress their loathing and contempt and become unable to acknowledge evil and injustice. Then slowly the process of self-justification grows in them; under the terror of evil they learn to justify and accept evil. Inwardly they build up all kinds of theories why they accept evil. However, when the terror disappears, and they can breathe freely again, the paradoxical justification may finally be corrected. It is dismaying to concede the dilemma we find ourselves in—that we need outer and inner freedom in order to become good and just.

Chronic Dangers

Modern psychology emphasizes the fact that there are constant small dangers, that there are accumulations of small pin pricks, often more dangerous than sudden attack. The recognition that there are such continual stresses sometimes quite near, other times far away, that there is no security, no freedom from defense can be tolerated only by rather strong personalities. Man is the only animal with complicated psychological conflicts. Born helpless, dependent upon his parents, having no innate neurological patterns which prepare him to react usefully, man, the two-legged, upright-walking and speaking animal, has the choice between various patterns of action. Hence he hesitates, is confused, delays and anticipates.

The by-word of the dictator that "life has to be dangerous" violates common psychological experience. The human being craves security. Well-measured dangers may stimulate our capacities and defenses, but man cannot always live on the frontiers of danger.

Constant danger and tension make the mind a rigid Maginot Line, an ever-alert fortress. Instead of useful defense, increased sensitivity may be built up. Modern psychophysiology teaches us that the organism reacts in different ways to acute sudden danger or to chronic dangers. Sometimes the chronic danger reaction is completely paradoxical. Where an acute danger, for instance, incites the stomach to decrease its production of hydrochloric acid and gastric juice, chronic danger and fearful anticipation may cause an increase in the secretions. In consecutive therapy we often experience that the chronic alert has to be changed into an acute alert. The chronic tension of the cold-handed man, for instance, has to be changed into acute warm and perspiring hands as symptoms of acute anxiety. From now on, the sympathetic system, as part of a general defensive system, may be able to react acutely, and find more useful adaptations, though many patients dread such activation of symptoms at first. The fear of the acute mobilization of the body is felt by people as something true and real, as something very closely associated with the body; the anxiety of the chronic alert, however, is something that is felt as not belonging to the emotional life.

The science of chronic mental pin pricks is just developing. We are now more aware of what an unconsciously aggressive mother may do to the baby, or what a latent psychotic can do to his or her mate.

Small quantities of mental poison, acting continuously on the human mind, may pile up, and the sum of the frustrations constitute a cumulative danger. Back-biting, slandering, expressions of envy, small acts of injustice, small unsolved quarrels can all have this cumulative effect. These mental pin pricks make us only slightly ill in the beginning, but in the end they cause penetrating wounds. This phenomenon is part of the pathology of everyday life and is, consequently, extremely important for our morale. Intense feelings of pain and hatred are aroused when people feel themselves misjudged, or when they fail in their own eyes. Such disgruntled people may come together on the basis of their common resentment in order

to form new aggressive social formations, such as the criminal gang or the political aggressors.

Actual psychopathology has pointed at many other forms of mental danger, which sometimes take the shape of acute conflicts, but more often the chronic accumulation of pin pricks. The dangerous action may begin in infancy, for instance, as continual rejection and exposure to too much insecurity. Later, unsatisfied sexual drives may have a continually disturbing impact or result in a fight against hypocrisy and archaic taboos. Any one of these accumulated small causes may suddenly lead to an outburst if they have been nursed and stored up inside the psyche. Often we find that a small stress becomes a token or trigger for the outcropping of the neurosis. This is what happens for the most part in battle neurosis. Fear and stress of war suddenly activate long-hidden conflicts.

Change and Abstention as Dangers

Abstention, withdrawal, starvation, deprivation, or change of habits may constitute acute danger to the organism which can be of clinical importance, according to the severity of the resulting symptoms, such as epileptic equivalents and psychotic episodes. In all such cases we may speak of a disrupted equilibrium and breakdown of automatic adaptation and feedback.

Psychologically it is important that we are aware, through outward signs, neither of the degree of potential adaptation which people possess nor of their hidden faltering adjustments.

Even the cessation of outside danger can bring manifold new reactions to the fore comparable with acute danger-reactions. Now people can let themselves go, and all the repressed anxiety and fear comes out into the open. Examples of this paradoxical outbreak of symptoms are the panics in Napoleon's armies after the victory of Jena in 1807, and the collective nervous breakdown in Dover in 1944 after the sudden cessation of the daily shelling to which the people had been subjected for four years.

During the Second World War we could experience how the rumination of the catastrophic experience in itself could become an anxiety-provoking experience. Many of the acute anxiety states related to war and invasion broke out after an incubation of months.

To this subject of the psychology of pathogenic anticipation and rumination belongs also the noxious influence sudden change in food habits and other social habits may have on people. The psychology of the immigrant and even of the summer traveller is fraught with these dangers.

Technique as Danger

A survey of concepts of danger would be incomplete without mentioning the paradoxical effect of our technical tools, which were originally meant to help man in his struggle for life.

Technique is born of a biological shortage. Man's infantile dependency and lack of adequate biological tools may have sharpened his intelligence and creativity. As a result, he invented technical tools as substitutes, by means of which his life became easier and more secure. He has become more independent of the dangers and threats of nature.

But these same techniques have also turned against him. Man has created unemployment with its concomitant feeling of rejection, as well as the perfidious tools of war enhancing his aggressive power. The useful machine has excluded many people from the process of production and has made it possible to concentrate productive power in a few hands. Scientific technique may put dangerous destructive powers in the hands of incompetents —automobiles in the hands of morons and alcoholics, atomic bombs in the hands of political adventurers.

The growth of technique and the luxuries it offers have confused our immediate mental needs. Flight into technical security takes away the training and strengthening action afforded by minor stresses and thus may increase our sensitivity. In the metropolis there is practically no challenge to face the forces of nature outside us and the forces of instinct inside us.

However, the intrusion of mechanical concepts into the human mind has other consequences for the internal growth of our defenses. Too much intrusion, resulting in too much

training and conditioning and restriction, may force man to become a mechanical tool instead of a living self. Pathology calls these internally automatized people compulsives. But every member of the human race is more or less burdened with the same tendency to automatization. Spontaneous self-expression grows more and more fraught with danger in a world where technique suggests ever more conformity of behavior.

In pathology we find the results of overburdening by technique and civilized habits described as the *effort-syndrome*. In such cases every effort beyond the every day mechanical duties produces some organic symptom, related to an old infantile fear of spontaneity. Changes in behavioral pattern are almost impossible today. New adjustments have to be paid for with disturbances of the central regulating sympathetic system. The price for such intrusion is for the most part a rigid, mechanical conditioning, arousing fear and disintegration when a new unknown stimulus or danger threatens the organism. From an unexpected source we find illustrated the neuroticizing action of daily overburdening of the mind as it is enhanced by special regimens or social habits. Dr. George Nasteo describes in a communist paper in Bulgaria (Bulletin: Free Europe Press, Nov. 29, 1955) how the party bureaucrats are creating new cases of neurotic illness among workers and intellectuals. Extra long working hours, the duty to attend over-long meetings afterwards, bad working conditions, red tape and bureaucratic pressure are the causes of a large number of nervous disorders. They cause the deepening of unresolved conflicts between people. Unfounded and unjustified accusations also increase the number of cases of neuroses.

The world will some day witness a tremendous battle between technology and psychology, between the dictates of mental mechanics and the clarified knowledge about human emotions. In a purely technical world, everybody would become a number on a card index, covered with labels and diplomas, but nevertheless a passive part of a metric system. By comparison with the might of machines, people now feel their smallness, weakness and inferiority even more—and at the same time man acquires a delusion of power, an omnipotence he is unfit to use. Think, for instance, of the raving frenzy and fanatical driving along the autostrada. People forget the power of the mind. It is a fallacy to think that ours is only a technical and industrial revolution. Moral evolution is more important. Can man cope with the destructive toys he has created?

Every civilization, as far back as the ancient ones, has tried, after a period of free development, to stabilize itself into rigid technicized forms. As a result, the veneration of technique and technical systems has turned knowledge into power instead of wisdom. The mechanical instruments of destruction have taken over our not yet controlled animal urges toward destruction. By now the destructive potential of weapons is so great that we can all become easy prey to those who yearn for absolute power. And this is the human and psychological side of the problem of danger because excess of technique in hands of inadequate minds eventually may destroy mankind.

Immunization against Mental Danger

Following the general biological rules of behavior—as we saw already in the traumatic experience—the human organism may react to danger in one of two ways. Frequently, both reactions occur in one individual. After a general alarm reaction there is first the immunization reaction, comparable with Selye's resistance reaction. Every successive stimulus or stress encounters more resistance and ultimately loses its quality of dangerous impact. In this case we may speak of active mental defense or resistance. Life forms its own mental and physical antidotes. Secondly, there is the passive reaction. Here the psychosomatic organism becomes more and more sensitive to the dangers acting on it. We may compare this form of reaction to the allergic reaction as a result of the formation of allergic products in the organism. Actually, it is impossible to separate the somatic and the mental allergic reactions. There is a continual relationship between the two. People with allergic reactions, such as asthma, often react hypersensitively to mental trauma also. Beside this we find that physical allergies can be influenced successfully by psychotherapy. Pride, fear, passive retreat and over-dependence are all well-known mental factors in

allergic disease, and, the other way around, allergy seems to form a predisposition for fear and anxiety.

In the process of immunization and allergization (sensibilization) we recognize too the principles of attack and flight with which we are familiar in several mental defense reactions. Increased cardiovascular reactions—higher pulse rate—often are the first symptoms of increased adaptation need for the organism. The organism senses danger, and mobilization of defenses starts—the motor accelerates. The subject experiences increased tension and apprehension. Everybody may experience this after simply overeating, or after drinking too much alcohol. However, the same mobilization may take place when there is merely a fantasy or anticipation of danger.

All this becomes even more complicated when we take into account the mutual interreactions between fantasy, fearful anticipation and increasing allergy toward stimuli from the outside world. For our purpose it is enough to be aware of the fact that there is an intimate relationship between danger, anticipation, and allergy.

Resuming our line of thinking we may conclude that *the concept of danger* is a very ambiguous one, dependent not only on the malicious intrusion of noxious influences from the outside world, but also on the internal build-up of resistances and defenses in the person. Only by understanding the alarm and fear reaction in man more and more clearly do we learn what physical and mental dangers are. Yet, it is also important to realize that in the processes of anticipation and the building up of fantasy, alarm and fear may have become secondary dangers, often worse than the primary danger. The resulting repression and frustration have become new dangers. Man often suffers more through the inner danger of his own failing defenses and mobilizations than through the reality of outer danger.

The Semantics of Fear and Anxiety

Every student of the concept of fear who considers the subject from a philosophical point wonders whether fear is a prime factor in man's nature without any explicable cause. Is fear in man connected with his innermost biological entity? Is being alive the equivalent of being continually in danger? Is man's psyche really built on fear and hope only? Or they ask other questions such as: Is one and the same fear behind all apparent motives for the fear reactions?

There is a general tendency to answer the last question affirmatively, although psychiatry, even when it differentiates between various aspects of reactions to danger, such as anxiety and fear, goes on to use these words and concepts without much semantic awareness. This is true for analytic as well as nonanalytic literature. Let us, therefore, go further into the verbal expression of *fear* itself. The word fear conveys a variety of experiences and emotions. Fear is related to the general Sanskrit form *per*—to go through the experience that causes the feeling—as we find back in ferry and fare. Later it acquired a narrower meaning: going through a disaster or calamity; in Old English: *fare, affeared, afraid.* The German *Gefahr*, and the Old German *faer* meant sudden peril of travelling, of separation.

Anguish comes from ange: pain: achos: ache. Awe is the same word. The Dutch and German word *Angst* (angoisse—anxiety), *inge*, etymologically means the narrow path or the narrow escape, again derived from anguish: angustus; narrowing. Another derivative is from anxus, angere: to throttle, choke, cause pain. Dread comes from the Sanskrit *draedan*: to shake. The French word *craindre*: to fear, originates from the Celtic word cretno, meaning to suspect or surmise and from the Latin word tremere, meaning to shudder, to shake with fear. Panic is derived from mythology: it is the fright and terror caused by the fearful sight of the ugly god Pan, and hearing his terrifying noises. Although originally used as a word expressing collective fear, it is now also used to indicate a state of panic in the individual. The wild shepherd god Pan was also a god of universal power and of the unknown dangers of darkness. Horned, long-eared, and half goat, half man, Pan symbolizes the fight between animal instinct and human control.

Awareness of time and anticipation increases the potentialities of danger. Our concept of compulsiveness as defense against fear was already expressed in old verbal cues. The latin word *meticulous* for fear, coming from *metus*,

full of little fears, has gradually acquired the meaning of being excessive careful. It is also related to *timid*, *temere*, to be afraid and probably derived from the Sanskrit *tam*, being breathless, being nearly choked, also being in darkness. Language tends to make a distinction between the vague anticipation of a threat to life and the clear actual perception of danger. Thus, in English, dread is a blind and vague feeling, while fear generally implies an immediate, known cause. There are students who prefer to reserve the word *fear* for the well-circumscribed feelings in relation to known danger (Freud's true anxiety) and the word *anxiety* for the vague feelings related to repressed, unconscious dangers (Freud's neurotic anxiety). However, clinically, both expressions are used in the same sense. And what is more important to the clinical user of the word, unconscious and conscious concepts of danger are *always* connected with each other. The unconscious fantasy determines what will be accepted or repressed as external or internal danger. Linguistically speaking, fear and anxiety are always intermingled and partly conscious, partly unconscious. In analogy to the feelings of danger of which man is partly aware, we may speak of protofear or preverbal anxiety in infancy in these early phases of life when there is as yet no communicating ego or conscious awareness, but the biological alarm is already in action.

Some philosophers, Kierkegaard (9) for example, identify fear with existence on this earth. For them it is the horror of the insecurity of life, which every human being must learn to endure. Fear is, for Kierkegaard, a typical manifestation of man's puny position. It indicates the limitation of man. This explains why fear is always connected with a feeling of inferiority, of being hopelessly outstripped. Janet (8), the French psychologist, is of the opinion that the sensation of fear always indicates the presence of something threatening the deepest elements of life; all fear is fear of the possible approach of death.

But for many a thinker, fear also includes fear of the unknown; it is a primitive and instinctive defense post from which we try to resist the continual intellectual exploration and progress of life. It operates in the service of dependent security, but not in the service of freedom. Fear in itself is a defense, an alarm signal, an anticipation and mobilization. Primarily it is the instinctive desire either to avoid danger or to face it adequately. That is why we encounter the somatic mobilization patterns of fear in all animals.

The same is true for the normal human being. Nearly everyone admits to have or to have had at one time some fear or apprehension in which the fear of darkness and of loneliness plays an important role. There is also a fear of not being liked, or not being smart enough, or the fear of not being able to cope with one's own ambition.

No one can completely resist his own defensive anticipations, however much he dreads showing fear. Both vague anxiety and conscious fear are expressions of the instinct of self-preservation. Thus we may say that instinctive fear provides a great advantage in the struggle for life; it provides us with useful warnings. Homo sapiens in particular needs instinctive fear because he is an animal poorly equipped physically for defense. Man, the organically immature, dependent fetalized being, relies more than any other animal on his inner warning systems. The danger may be real or imaginary, inside the body or coming from the environment; only a warning system that is intact and alert stimulates him to evade or conquer danger.

Fear mobilizes man. As Kierkegaard says: "It is a school," it puts man on the alert—against life, against death, against danger, against growth. Fear may have a stabilizing and protective effect although the excitement of fear can replace the instinctual warning system. Love of fear may be preferred to the satisfaction of the drive. Fear calls to arms various defensive reactions. However, too much defense may in itself become a danger. Man then suffers under his own alarm, his own fears. There is even a fear that kills; the asset of instinctive protection has become a liability.

These are in short the primary associations to the usage of the words fear and anxiety. Being aware of the semantic problem involved in the concepts protofear, fear, and anxiety, we learn to better understand the words and the paradoxical ways in which we use them.

Fear is an ambivalent concept. Though the words are used mostly in a negative sense—as unpleasant feelings—both fear and anxiety imply the necessary mobilization of defensive forces. In therapy we often have to arouse anxiety in order to bring to life long dried-up mental and organic sources of defense. Anxiety is often the first indication of the change from chronic repressed defense into an acute state of alertness. Linguistic, psychoanalytic, and hormonal concepts of fear and anxiety meet each other in these statements.

Before giving a survey of the semantic varieties of fear (table 1), I want to mention the manifold, partially synonymous words, all expressing fear yet never identical in mean-

TABLE 1.—SEMANTICS OF FEAR

Meaning	English	French	German
Unconscious recognition of danger Discomfort Irritability	stress reaction preverbal anxiety micropanic		
Biological defense Protofear Predisposition to anxiety	acute shock pain fright scare startle reaction automatic anxiety	frayeur	Schrecken, Schrecklicher Angst
Feeling of helplessness Feeling of paralysis Too weak to cope with danger; Surrender to fate	fear fright dread horror and perplexity	peur (La peur aveugle)	Angst or Furcht
Impending evil	dread free floating anxiety	peur intense	frei flotierende Angst, innere Unruhe
Anticipation of unknown danger Fearful expectation Painful uneasiness of mind	perpetual restlessness insecurity phobia latent panic signal anxiety	anxieté	same as above also: mir ist bange
Causing alarm Shock Narrow escape (angustiae)	panic anguish pang agony pavor nocturnus	angoisse	Todesangst, beklemmende Angst
Recognition of danger, but one is courageous Fear stimulates life	being scared fear stress adaptational conflict	crainte (la crainte raisonnée)	Furcht or Angst Befürchtung Scheu (related to taboo)
Awareness of superior power Insignificance in the presence of mysteries	awe dizziness of freedom (Kierkegaard) shock of growth (Goldstein)	respect reverentiel	Ehrfurcht (frommer Schauer) Gottesfurcht
Confrontation with the unknown Loss of norms and evaluations	being lost remorse pain cultural anxiety	repenter la nausée d'être pour soi, douleur	Reue, Schmerz Unlust

ing, such as dread, fright, scare, alarm, dismay, consternation, panic, anxiety, terror, horror, agony, concern, foreboding, misgiving, worry, anxiousness, uneasiness, apprehension, tension, self-torture, blue funk.

The Subjective Sensation in Danger

In man the dangerous conditions creating fear and anxiety are traceable to the physical and the psychological systems; in each of these spheres there can occur such a threat to physical and inner mental life that continued existence is restricted to progress along a very narrow path only. Being afraid means a slackening of elasticity in life. It is the opposite feeling from ecstasy, elation and intoxication, which are expansive and give a feeling of oneness and total identification with the universe. Fear ensnares the human being and prevents him from getting complete release from himself. He always remains mobilized, on the alert, on guard; and therefore he never lives in complete freedom. It is this fear that causes the craving for a particular illusion of freedom; to live without fear and anxiety.

Up to now the subjective experience in moments of danger—its phenomenology—has been studied least of all. What does the human being really experience? The sensations caused by danger produce such a limitation of consciousness and self-observation that the person who is afraid is no longer in a position to take stock of himself. Few people can give a coherent account of their sensations in panic. I have often been asked if I was really afraid when I was condemned to death. I do not exactly know. I always maintain that I was not, but my muscles were, and they carried me along to safety. When I ask war heroes about their fears, they laugh somewhat ironically and confess that they no longer remember just what part fear played in their actions.

The change in sensitivity and perception in the moment of danger is best illustrated by the following personal experience. Because of a sudden acuity of senses during the night I was transported as a prisoner of the Nazis, I had overheard their conversation with a French policeman who had come to fetch his own smugglers from prison. This settled my plan to play the role of a French money smuggler. When the French discovered their mistake, they were willing to help me and even advised me to jump on a train near their station prison. There I was out of the immediate threat of death, free on a train moving to Paris, but surrounded by hunting enemies. My mind was clearer than ever; I enjoyed the summer landscapes as never before; I exchanged clothes with fellow travelers on this labor train and changed my outlook completely. I became friendly with a mother and her baby, and with the crying baby on my arm, quasi-quarreling with the mother, I went through the inspecting German cordon at the Gare du Nord.

Now I was free! I took the subway, and then hell broke loose. In a frenzy I found myself travelling all day long in a Paris subway as if I were still escaping my enemies. I trusted nobody. I could not remember any addresses of friends in Paris. I changed trains till it became dark. Then I went to a barber shop to have a wash and a shave, and only then did I find myself again.

Such so-called depersonalization feelings are repeatedly described in all kinds of war literature. Later I remembered so very well my deep feelings of guilt at having escaped death and having left so many of our prison group on transport in the hands of the Nazis. Something in me had already surrendered to death. Yet something more vital had saved me. Later, walking through occupied France in order to reach non-occupied territory, I was even more taken by that ecstasy of living, of absorbing reality through all the senses.

In this whole process of realization of what to do, what you are, what you want, small symbolic actions are of such tremendous importance. The change of clothes (in order to look like other Frenchmen), the wash and shave, all helped to give me a valuable new status. The deflated ego of the prisoner in utter danger became an ego again. The senses are activated in order to build up the new ego. A new emotional investment in the reality starts (new object cathexis).

Later on, while directing welfare work in the Allied armies, I learned much more to realize how valuable clothes and special foods are in

order to build up the deflated egos of those who have lived under pressure, fear, and humiliation.

Others who worked under the same panicky circumstances describe the same type of depersonalization feelings. Cohen (3) mentions three stages in the experiences of the prisoner. First, there is the initial reaction with its feelings of depersonalization, even when the prisoner's conception is more or less in line with the real situation. Second, there is the stage of adaptation. Many of those in acute depersonalization were able to find new adjustments, but those prisoners who remained possessed by fear and fright became more and more apathetic, and let themselves go more and more. In all of them a certain degree of regression had to take place because of the inhuman, nearly animal circumstances. During the third stage of acquiescence, the prisoner became settled and even regained several aggressive activities, as we know from the underground movement in the camps. The second phase was the most difficult; those drowning gradually in their apathy, submitted to death and gas chamber, hardly aware of what was happening to them. In their fantasy and apathy they had completely retreated from danger.

People who adjusted themselves best to those dire circumstances were those guided by strong religious or philosophical principles, all those who were able to master their fearful anticipations.

In several of them the repressed expression of fear came back after the liberation, when they were allowed to let their anxieties take possession of them again. One of my patients, a compulsive hypochondriac, lost his neurotic preoccupations completely during his two years' sojourn in a concentration camp. But after the liberation he went through a nearly psychotic cataleptic state, after which he recovered completely from his ancient and actual fears.

Some patients maintain that fear is located somewhere in the stomach or in the legs, like something heavy lying there. Fear located around the heart is well known; it is related to the rhythmic activities of heart and life, or it is experienced as a constricting pain. I know a woman who always feels fear in a post-operative scar. Some people refer to remarkable optical phenomena, flickering, and colored circles of light. Several epileptics describe their fear as a deep red color. Delirious people see myriads of small frightful animals on the wall. The feverish patient experiences his fear as constant pressure, as of mountains heaped upon him. Others see fire approaching them or fancy they are being pursued by black men. Analytically speaking we may relate some of these fears to revived reminiscences of the birth trauma.

The way danger and fear are experienced—acutely or chronically—expresses reminiscences of old archaic dangers, as a rule. There is often a feeling of suffocation, as if somebody were catching you by the throat (globus hystericus), there is gooseflesh as a remnant of an archaic pylomotoric reflex and camouflage. The mouth is dry, the skin perspires profusely, the cardiac motor works at high speed. The body attempts to throw off all that tries to invade it; vomiting and diarrhea often occur. In order to get rid of tension, frequent urination takes place. All the ancient dangers of toilet training become rampant. Or danger is experienced as a tremendous cold; people tremble and shudder. In others, long repressed sexual yearnings awaken again and ask for a last fling before the expected moment of doom and death. Yet all this can go together with a complete conscious denial of danger and fear. (A more elaborate and systematic study of primitive responses to danger will be presented in a future paper.)

In extreme danger, the entire apparatus of perception has changed. Our subtle warning signals do not work. In panic we no longer feel pain. Our skin becomes insensitive. Our visual perception changes. Our hearing becomes over-allergic. The normal after-images of the eye, with the often contrasting colors, are interpreted as ghosts or persecutors. Consequently, in darkness we see the white contrasting images but interpret them as phantoms or as representations of the enemy.

Only through hypnoanalysis or psychoanalysis can the particulars of what is going on during a fear attack be brought to the fore. In the treatment those revived reminiscences

often provoke the same anxiety spells. Birth trauma, early oral conflicts and early instinctual frustrations play the most prominent role in those reminiscences.

Fear is in itself a contagious emotion. The human power of imagination often falls short of being aware of it, but the reaction is there, nevertheless, as for example, in cases of mass panic. Hours, sometimes days, afterwards persons are still pale, and the psychosomatic reactions continue to work within them.

Each person experiences fear in his own way and often without much awareness of it. We are often more conscious of the psychodynamics by which fear is suppressed, assimilated and overcome, hence my insistence on discussing this descriptive aspect of the matter.

In ultimate danger, in the few moments before the calamity, the mind may turn to an ecstatic reviewing of one's life history. Many victims of drowning or mountain accidents who were saved give witness of these ecstatic moments, covering all sorrow and distress of the actuality. Eternity is seen in one second; the unconscious knows no time. The ecstatic fantasy protects against the catastrophic reality. The awareness of danger is annihilated by the autonomy of man's mind.

Resumé

In an attempt to bridge the various meanings included in the concepts of danger, stress and fear, a semantic and descriptive approach is used in order to link the phenomenological method with the clinical and psychodynamic concepts.

In the course of such an inquiry several antimonies are encountered: acute danger as against chronic danger, general danger as against specific (symbolic) danger, internal danger as against external danger, fantasy danger as against real danger, anticipation as against rumination of danger, strain as against stress, physical danger as against mental danger.

The study leads to several surveys, illuminating the relation between danger and various stresses, another tries to emphasize the manifold, often confused, usage of the words fear and anxiety. Finally, the author illustrates with some personal experiences how unexplored is the subjective "rumination" of the biological response to danger.

BIBLIOGRAPHY

1. Bayley, H.: *The Lost Language of Symbolism.* New York: Barnes and Noble, 1952.
2. Cannon, W. B.: *Bodily Changes in Pain, Hunger, Fear and Rage.* New York: D. Appleton and Co., 1929.
3. Cohen, E. A.: *Human Behavior in the Concentration Camp.* New York: W. W. Norton, 1953.
4. Freud, A.: *The Ego and the Mechanisms of Defense.* New York: International Univ. Press, 1946.
5. Freud, S.: *The Problem of Anxiety.* New York: W. W. Norton, 1936.
6. Goldstein, K.: *Human Nature.* Cambridge: Harvard University Press, 1947.
7. Greenacre, P.: The predisposition to anxiety. *Psychoanal. Quart., 10:* 1941.
8. Janet, P.: *De l'Angoisse a l'Extase.* Paris: Alcan, 1926.
9. Kierkegaard, S.: *The Concept of Dread.* Princeton, 1944.
10. Korzybski, A.: *Science and Sanity.* Lancaster, Pa.: The Science Press, 1933.
11. Loosli-Usteri, M.: *De l'anxieté enfantine.* Berne: Huber, 1943.
12. Meerloo, J. A. M.: Abstention, rebound and readjustment. *J. Nerv. & Ment. Dis., 120:* 1954.
13. Meerloo, J. A. M., and Klauber, L. D.: Clinical significance of starvation and oral deprivation. *Psychosom. Med., 14:* 1952.
14. Meerloo, J. A. M.: *Patterns of Panic.* New York: International Univ. Press, 1950.
15. Meerloo, J. A. M.: The passive surrender to fear. *Nethl. T.vG.,* 1929.
16. Meerloo, J. A. M.: Shock, a psychosomatic phenomenon. *J. Nerv. & Ment. Dis., 111:* 1950.
17. Meerloo, J. A. M.: *The Two Faces of Man.* New York: International Univ. Press, 1955.
18. Russel, R. W.: Behaviour under stress. *Internat. J. Psychoanal., 34:* 1953.
19. Selye, H.: *The Story of the Adaptation Syndrome.* Montreal: Medical Publisher, 1952.
20. Shipley, J. T.: *Dictionary of Word Origins.* New York: Philosophical Library, 1952.
21. Simpson, G. G.: *The Meaning of Evolution.* New York: Mentor Books, 1951.

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THE COMPLEX OF FRUSTRATION[1]

A New Interpretation

NINA BULL AND EDWARD STRONGIN

In a recent publication (2) we have presented *goal orientation* as a biological norm of human and animal behavior, and we have posited a certain basic change in neuromuscular organization which occurs when the goal-oriented organism is interrupted in its forward course of action. We have called this change *frustration* and considered it as essentially a disruption of the forward-facing attitude which constitutes a postural preparation for activity directed toward a goal. This forward-facing, forward-looking attitude not only prepares for goal-directed action, but also maintains it. It can be profitably equated with the *reflex of purpose*, which Pavlov (6) has extolled as "the most important factor of life," and thus by inference placed above all the other reflexes described by him.

We have already written on the combination of aim and forward movement that forms the basic neuromuscular mechanism in goal-oriented behavior, at the same time presenting evidence that *aim* is predominantly visual in character and that interference with goal orientation shows up in visual behavior as the breakdown of binocular coordination (2, 7). We are now proposing to approach the subject of interference from a very different angle, in order to clarify and also to elaborate the *psychological* conception of frustration as it applies to the interlocking problems of theory and therapy.

The term frustration (from *frustra*, Latin for *in vain*) will be used, as is customary, to cover both the tensional state and the resultant feelings of being blocked in an endeavor. This is in line with—and a development of—Attitude Theory (1), which postulates *all feeling* as generated by tensional states considered under the general heading of *motor attitude*. We are now proposing to analyze this particular tensional state in order to expose its complex yet basically consistent structure; for it is definitely not a random breakdown that confronts us in frustration.

In its least complicated and most familiar form frustration connotes the interruption of a project by an *obstacle;* that is to say, it occurs when the organism encounters more outside interference with its progress toward the goal than can be "taken in its stride." Under these conditions, of too much interference, the original type of directed activity ceases, the goal being partially eclipsed or obscured entirely, while the main preoccupation is now with interruption itself and/or the interrupting obstacle.

It is clear that concern of this kind, amounting to *fixation on the obstacle* with its disturbing effects, would represent a shift from the original positive orientation, which is still preserved in part, however, through maintenance of some elements of the tensional set. The very presence of frustration indicates that the project is not entirely abandoned, that the goal is still desirable; and the significance of any obstacle (qua obstacle) is relative to this important fact.

Thus we are dealing first and foremost with a special type of conflict between two orienting attitudes which are related but, for various reasons, incompatible—the first one aiming toward a goal, and still operative, though impaired; the second one initiated by the obstacle, and aimed at getting rid of it. According to the present thesis, the essential feature in frustration is just this special type of conflict, based on the interruption of a goal-directed course of action sufficient in its impact to bring about a functional disruption of the forward-facing attitude, and temporarily preventing a reestablishment of the positive instinctive drive required to achieve the goal.

The striking contrast between goal-oriented and frustrational behavior, which is stressed

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FIG. 1

FIG. 2

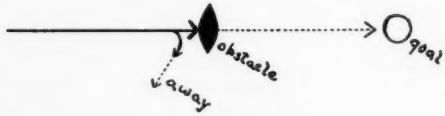

FIG. 3

repeatedly by Maier in his study of frustration (5), can now be logically approached in terms of the above conception. Goal-oriented behavior is essentially a unified approach, with one objective. It has a single aim and can be represented by an arrow (fig. 1).

Frustration, on the other hand, starts out with two objectives—goal and obstacle. Its onset can be roughly represented in the accompanying diagram, although even the initial reaction to obstruction is not so simple in its orientation as this diagram would indicate (fig. 2).

In the frustrational situation there are these important points to be continually held in mind: the obstacle is always *nearer* than the goal, looms *larger*, and requires to be dealt with *first*. Under certain favorable conditions the interruption will be seen in proper perspective, that is, in its relation to the more distant goal—lined up with the original direction, and taken like a hurdle if it cannot be avoided. This is what happens very often in the appetitive stage of goal-directed action, which is geared for flexibility in seeking and pursuing (2), and even to some extent for overcoming obstacles. But where for any reason the interruption is too threatening, it is reacted to as an emergency—that is to say, *with fear*—and its alignment with the goal becomes impossible. Actually there is always a double orientation tendency initiated by obstruction: (a) to fixate on the obstacle, preparatory for attack, and (b) to turn away (avert) from it, a variety of factors combining to determine the relative strength of these two riddance tendencies in any given situation. Thus the organism may attack the obstacle head-on[2] (see figure 2) or turn away and find itself distraught both as to direction and intention. In either case the primary conflict, based on the interruption of directed behavior, turns out to be the starting point for complication and distraction, as shown in figure 3.

This diagram shows three distinct but interlocking action-tendencies—one of them still directed toward the (more distant) goal, the others aiming *at* and *from* the (nearer) obstacle, respectively. We propose, for reasons of precision, to limit the term frustration to situations where interruption takes on this character of *threat* and automatically produces the mixed type of emergency reaction which is found in all "emotional" response to danger. Thus all frustration would definitely connote the presence of fear, which is not, however, sufficiently overwhelming to cause a complete disappearance of the original goal-oriented tendency. It also connotes the presence of *dislike*, a gut-reaction with olfactory beginnings (1, pp. 107–113), which reinforces fear in its compulsive efforts to rid the organism of its trouble. This does not mean that the obstacle (as the source of trouble) is necessarily offensive in itself, for *anything* can be an obstacle, even potential "good" which turns to "bad" simply through causing interference and disruption.

The schema in figure 3 is offered tentatively as a basic complex of orienting attitudes that is common to all frustration, and has the advantage, in spite of being over-simplified, of furnishing a point of departure for further exploration and analysis.

An important subject that becomes accessible through this operational approach is the self-preoccupation which follows or accompanies frustration and forms a secondary obstacle to progress. Not only does frustration spell danger to the project that is being inter-

[2] While the direct attack is going on, fear is not felt, nor frustration experienced, the riddance impulse being strong enough to unleash an emergency reaction of full-blown anger at the obstacle. But since anger at the obstacle is rarely a successful way of dealing with obstruction, the result is likely not to prevent frustration, as intended, but merely to postpone it.

rupted, and all that this involves, but the breakdown process that underlies frustration constitutes a serious danger to the organism itself, now threatened by loss of its physiological integrity (resulting in discomfort) and by its incapacity to recover, resulting in a sense of helplessness. Right here we have the point of entry for further complication—the downward path into *depression*—where the organism, having lost its goal, becomes fixated on some aspect of its own entanglement and consequent discomfort, while it perseverates in a display of helplessness and protest, designed, like screaming in a baby, to call attention to its plight. And while the focus of attention may shift from point to point, it always includes the factor of *incapacity* caused by the interruption of progress toward a goal. Thus depression represents continuing frustration, in which we find a secondary phase resulting from and added to the primary one.

The fall of man—for such it actually is in terms of nervous integration—from the primary reaction to obstruction (as shown in figure 3) into depression, with its low-grade selfward orientation and its muted expectancy of succor, is here considered as stemming from the original impulse to cry for help in every kind of trouble. This impulse, which can perseverate indefinitely in its preparatory form of motor attitude, is chief among the various regressive patterns that characterize adult depression according to the present thesis. In a sense the crying pattern, its early violence toned down, holds the key to all depression, from melancholia to everyday unhappy moods in ordinary people, these latter being nothing but depression in a milder form.

Our thesis is that self-fixation in depression is a form of grievance over primary frustration and constitutes another even more perplexing obstacle to progress—this time *within the organism*. The selfward orientation[3] is in the nature of concern about the breakdown process in one or more of its many aspects and/or after-effects.

In line with the above conception, the terms frustration and depression can be considered as descriptive of two different aspects of the breakdown process—the process being actually continuous, with no real line of demarcation between what we have here suggested as the first and second stages. However, these two terms are definitely not synonymous, and the following distinction can be profitably made: frustration is concerned primarily with interrupted purpose, while depression is concerned primarily with the plight in which the organism finds itself as a result of its frustration and attendant disappointment. Thus, in frustration the aggressive riddance impulse is predominant, and obstacles appear on every side; while in depression the abandonment of independent effort is predominant, with resort to crying and/or various related methods of appeal for help.

The diagram in figure 3 is like a map in representing certain essential relationships that can be visualized in all frustration where organism, obstacle and goal are spatially distinct; that is to say, whenever the obstacle is located *between* the organism and its goal. This is the most familiar and readily apprehended form frustration takes, and we have concentrated on it here because it can be used to illustrate a principle that also holds for interference in its less obvious aspects. Thus, for example, the schema is applicable to depression, considered as an extension of frustration, with interference now located *in the organism*. This offers a reasonable explanation of certain seemingly unnatural impulses to attack and to escape from something in oneself, these being primary modes of dealing with an interference wherever it occurs. Also the schema can be used to clarify the common and exceedingly confusing situations where goal and obstacle are difficult to separate from one another.

Within the framework of the same conception, aggression in the hostile sense is open to interpretation as a primitive method for getting rid of interference; while the innumerable devices for "escape" (currently so-called) are seen as ways designed to free the organism from frustration by abandoning the situation as a whole—and throwing out the goal together with the obstacle.

A different type of situation presents itself

[3] This would seem related to the self-referent state which T. Burrow has called *dilention* and which he finds to be a defect common to the entire human race.

when interruption takes the negative form of disappearance, or lessening in value, of something that was depended on, something that was furnishing *support*.[4] But here too the principle holds good, and breakdown occurs whenever the loss is experienced as a threat.

There is another aspect of frustration which must be mentioned in passing, although it cannot be elaborated here. The organism, having already suffered severe frustration or repeated frustrations, along certain lines of its normal endeavor, becomes *inhibited from trying*—that is to say, experiences frustration and depression without having become goal-oriented first. This is a familiar meaning of the term "inhibited" and signifies a loss of normal initiative in certain—sometimes in all—directions. Many states that are spoken of as inhibition can be better understood and dealt with if the frustration that occurred on previous occasions is recognized as an underlying factor in the process.

This condition, of being *inhibited from trying*, is one of the various types of incomplete activity where failure to get going becomes itself an ill-defined frustration and cause of vague depression for which there seems to be no adequate explanation.

In view of all the foregoing discussion, we offer the concept that frustration is the fundamental problem in neurosis. This idea is not entirely a new one. Maier has suggested that neurosis is a relatively permanent state of frustration (5); and T. M. French, approaching the subject less directly, has found in every psychopathological manifestation a disintegration of the goal-seeking mechanism (3). "Frustration ends," he says, "with *physiological disintegration* of the pattern *of a goal-directed striving*" (4).

Even more important than the problems of disintegration and neurosis is the problem of everyday unhappiness, which, as we have earlier suggested, is depression in a milder form, and thus a product of frustration according to the present thesis. The unhappy person is concerned with what has happened to him—namely his own disruption—and is lamenting over it; and the lamentation or grieving process, which is a form of muted crying, tends to perseverate simply because expectancy of succor is the hope implicit in every gesture born of this reaction. In short, the unhappy person is self-fixated and depressed, and cannot regain his own initiative inasmuch as he remains expectant—waits for help or solace from outside sources and very often does not even know that he is waiting.

Seen from this angle, unhappiness becomes available for study, since much of the difficulty in understanding it has resulted from failure to recognize the sequence of events, and separate the element of self-concern not only from the complex of frustration which precedes it, but also from the innumerable impulses that follow it—designed to bring relief and/or concealment.

While many people learn to carry on and do effective work despite the hold-back of a continuing self-fixation and expectancy, this is by no means the best of which our human nature is capable. We are now investigating some possibilities of dealing directly with depression as a complex act instead of as a state, in order to prevent potential extension and expansion of the process into the far more complicated patterns of neurosis. We are also studying results obtained by interpreting conflict and confusion in terms of goals and obstacles, and thus introducing a *sense of direction* into what was a jumble of impulses, feelings, impressions and memories to begin with.

Regarded from the present point of view, a *sense of direction* is the key to order in the mind, its fullest, happiest development being found in such purposive activity as is headed toward a long or short-term goal in the form of a biological objective required to satisfy some basic need. This goal-directed purpose we have called *goal orientation*, as distinguished (a) from the various destructive forms of purpose, aimed at the obstacle and developed out of simple riddance-reactions, which keep the organism fixated on interference so that the goal is either pushed aside or temporarily forgotten; and (b) from the innumerable less conscious or wholly unconscious forms of purpose, where the goal has never been envisaged, and the individual is unaware of his own intentions.

[4] Tolman has pointed up the important fact that *supports*, of one kind or another, are essential to the attainment of *any* kind of goal (8, p. 85).

As cultural directives have modified and tended to distort our basic biological patterns, goal orientation in its inherited integrity has perforce become more of an ideal than an actuality. Too many of man's objectives are obstacles or means of escape—not goals at all, as we conceive the term; and even when this is not the case, the would-be goal-oriented attitudes and actions are largely hampered by unresolved frustration, so that two disparate action-systems are operating simultaneously.

Further research is now in progress to test some of the theses outlined in this and the preceding paper (2) and to determine whether interference can be established as a valid point of reference from which to study the various aspects of frustration and depression. This would make possible a scientific exploration of the contrasting modes of operation that are represented by psychological integration and its breakdown.

BIBLIOGRAPHY

1. Bull, N.: *The Attitude Theory of Emotion.* New York: Nerv. & Ment. Dis. Monograph No. 81, 1951.
2. Bull, N.: The mechanism of goal orientation and the manner of its disruption. *J. Nerv. & Ment. Dis., 122:* 42, 1955.
3. French, T. M.: Goal, mechanism and integrative field. *Psychosom. Med., 3:* 226, 1941.
4. French, T. M.: *The Integration of Behavior.* Volume I, Basic Postulates. Chicago: University of Chicago Press, 1952.
5. Maier, N. R. F.: *Frustration, The Study of Behavior Without a Goal.* New York: McGraw-Hill Book Company, 1949.
6. Pavlov, I. P.: *Lectures on Conditioned Reflexes.* New York: International Publishers Company, 1941.
7. Strongin, E. I., Strongin, J., and Bull, N.: Visual changes and affective states. *J. Nerv. & Ment. Dis., 119:* 344, 1954.
8. Tolman, E. C.: *Purposive Behavior, In Animals and Men.* Berkeley: University of California Press, 1951.

INVESTIGATION OF RELATIONSHIPS BETWEEN "PSYCHOMETRIC" AND "BIOLOGICAL" INTELLIGENCE[1]

RALPH M. REITAN, PH.D.

In 1947 Halstead (5) classified existing contributions toward understanding of intelligence into three categories: psychometric intelligence, clinical intelligence, and the neurological conceptions of intelligence. Very briefly reviewed, these concepts of intelligence may be characterized according to the settings from which they arose, their postulates concerning the essential nature of intelligence, and methods used for determining intelligence.

The psychometric concept of intelligence springs largely from investigative efforts of psychologists, many of whom have worked in a non-clinical environment. The essential nature of psychometric intelligence has been variously presumed to be "organizational ability", "judgment", "abstract thinking", "adequacy of adaptability to new situations", and many other characteristics. As Halstead indicates, these characteristics frequently are as difficult to define as intelligence itself. However, psychologists have devised standardized testing procedures which have the advantage of reliable quantitative scoring, and have proceeded to evaluate their results with respect to a variety of possibly and/or partially relevant criteria. These efforts have resulted in a large number of scales for evaluating intelligence, many of which yield an Intelligence Quotient.

Clinical intelligence represents a concept derived largely from the contributions of psychiatrists and psychoanalysts. Again, essential characteristics of intelligence have been postulated including "memory", "the ability to form associations", "abstract" as compared with "concrete" behavior, and others. Descriptions of intelligence by writers whose impressions have arisen from clinical psychiatry are generally oriented toward the effectiveness of an individual's behavior as a major criterion of intelligence.

Neurological conceptions of intelligence are associated primarily with the effects of experimental or clinical brain lesions in human beings and lower animals. Familiarity with contributors in this area (Flourens, Goltz, Loeb, Feuchtwanger, Lashley, Munk, von Monakow, Kleist, Hitzig, Flechsig, Franz, and Bianchi) indicates that one of the major problems has been that of attempting to define the role of brain function or dysfunction with respect to abilities and behavior. The significance of aphasia with respect to intellectual impairment and the question of localization of abilities within fairly well-delimited areas of the brain have been two of the primary problems considered.

While much has been learned of both practical and theoretical importance in these various investigations of intelligence, serious deficiencies exist in each. Although the Intelligence Quotient has been of great value in many respects, studies of its agreement with various validating criteria have often yielded comparatively unsatisfactory results. However, rigorous validating attempts have not even been possible with the other concepts of intelligence since standardized and quantified evaluating procedures have not been developed.

Differential estimates of intelligence, implying some degree of incompatibility in the three views, often occur in individual cases. Recently we have observed a patient who had an I.Q. nearly in the normal range although his left hemisphere had been largely removed as a result of a large glioblastoma multiforme. This patient was left-handed and had no aphasia postoperatively. In patients with residual dysphasia we have measured severely deficient I.Q.'s which clearly did not correspond with observations of intelligence in everyday situations. Disparities between estimates of psychometric and clinical intelligence are also frequently encountered. These are illustrated by patients with high I.Q.'s whose neurotic difficulties preclude satisfactory adjustments even to relatively simple everyday problems.

Halstead (5) has recently proposed the con-

[1] This study was supported in part by the James Whitcomb Riley Memorial Association.

cept of biological intelligence which he has defined briefly as follows (6):

> . . . what I mean by biological intelligence raises a difficult problem in brief communication. Some years ago Cannon used the title *The Wisdom of the Body* for one of his books. I do not believe that Cannon intended to omit the nervous system in his concept. In biological intelligence I am trying to direct attention to the "wisdom of a healthy nervous system." It is my belief that psychometric intelligence, as reflected by the I.Q., does not adequately indicate this "wisdom" of the healthy or of its alterations in the pathological nervous system. We have repeatedly found normal or superior I.Q.'s in neurosurgical patients lacking up to one-fourth of the total cerebrum following frontal lobectomy. Yet our measurements of biological intelligence indicate that these are not normal individuals. . . . Biological intelligence, we believe, with high adaptability, is the normal outcome of the functioning of a healthy nervous system.

While this definition throws some light on the term by stating a specific orientation toward a homeostatically modulated type of expression subserved by a healthy nervous system, it does not have an operational character. The question remains, "What about apparent ability differences in various individuals all of whom have healthy nervous systems?"

A definition of perhaps greater operational significance has been given by Halstead in his detailed elucidation of the four factors which seem to compose biological intelligence, according to the measuring instruments used (5). These factors were derived, through factor analysis, as the fundamental test-clusters which most parsimoniously account for the results obtained. They were named by Halstead as the Central Integrative Field factor (C), the Abstraction factor (A), the Power factor (P), and the Directional factor (D), and summarized in a recent paper (7). The C factor is defined as a "memory" factor, and ". . . serves as the stable framework of the 'familiar' against which 'new' experience is tested." "The A factor, or capacity for abstracting universals or rational concepts, seems to be a general property of the cerebral cortex in man that is maximized in the cortex of the prefrontal lobes." The P factor is interpreted as a "cerebral power" factor which seems to be the energy source behind intellectual function and which is suggested by preliminary evidence to be related to cerebral metabolism. Finally, the D factor represents the ". . . avenue or modality through which intelligence is exteriorized in any given situation." These avenues may be selectively impaired as is seen in aphasic patients.

Halstead's results (5) indicate that psychometric intelligence as measured by an I.Q. test loads most heavily on the C (memory) factor of biological intelligence. Although Halstead used the Hemnon-Nelson Test for estimating I.Q., the Wechsler-Bellevue Scale has the advantage of individual administration, more extensive coverage of abilities, and more comprehensive normative data. Independent factor analyses of the Wechsler-Bellevue (1, 2, 3) indicate striking agreement in the factors extracted even though groups have differed with respect to age and diagnosis. Birren (2) concludes that "stored information" is the most important component in determining individual differences in scores on the Wechsler scale. "Stored information" would seem closely akin to "memory" as used by Halstead, and the hypothesis therefore exists that psychometric and biological intelligence may be more closely related than previously recognized. The present study was designed to determine the extent to which psychometric and biological intelligence may overlap or stand as separate entities.

Subjects

Fifty patients with proved brain damage composed one group, and 50 persons without evidence of past or present brain damage or disease composed the other. The subjects from the two groups were individually matched in pairs on the basis of color, sex, chronological age, and years of formal education in an effort to control irrelevant variance. A substantial amount of comparative information is present for these groups (9, 10, 11, 12), and they have previously been described in detail (9).

Procedure

The 10 neuropsychological tests contributing to the Halstead Impairment Index and the Wechsler-Bellevue Scale (Form I) were individually administered to each patient. The tests were administered and scored prior to receiving diagnostic information on the subjects and before the groups were composed.

Pearson product-moment coefficients of correlation were calculated for each possible pair of variables for each group. The coefficients were transformed to Fisher's z values, the standard error of the difference between z's obtained, and the significance of differences in z for the two groups determined for each pair of variables (8).

Results

Intercorrelations between the Wechsler-Bellevue variables for the groups with and without brain damage are presented in table 1. The median correlations in table 1 for the groups with and without brain damage are .60 and .59, respectively. Eighty-four of 91 coefficients were significant beyond the .05 level for the brain-damaged group and 90 of 91 for the group without brain damage. This indicates that the W-B variables have a substantial interrelationship regardless of whether or not brain damage is present. In testing the significance of differences in individual pairs of coefficients in the two groups, only 2 of the 91 pairs reached significance at the .05 level. These could easily have occurred by chance. Birren's (2) matrix, which does not include the three I.Q. variables, has a median correlation of .54. The medians in table 1 with these variables omitted are .52 for both groups. The present results agree closely with Birren's findings and indicate that the interrelationships between the W-B variables are approximately the same in a group with heterogeneous brain damage and a group having no brain damage.

Table 2 presents the intercorrelations between Halstead's tests for each group. The median coefficient for the brain-damaged group is .16 and, for the group without brain damage, .24. Eighteen of 55 coefficients were significant beyond the .05 level in the brain-

TABLE 1.—Intercorrelations between Wechsler-Bellevue Variables

Brain Damage	1	2	3	4	5	6	7	8	9	10	11	12	13
1. VIQ													
2. PIQ	.72												
3. F-S IQ	.93	.92											
4. Inf	.79	.62	.77										
5. Comp	.79	.61	.75	.66									
6. Dig Span	.78	.48	.69	.49	.39								
7. Arith	.82	.54	.75	.60	.53	.58							
8. Simil	.85	.65	.81	.61	.66	.68	.55						
9. Vocab	.81	.65	.79	.79	.69	.56	.55	.78					
10. P Arr	.49	.68	.62	.47	.37	.41	.38	.46	.49				
11. P Comp	.59	.74	.71	.61	.55	.34	.51	.65	.67	.53			
12. Bl Design	.63	.86	.80	.46	.46	.52	.60	.51	.40	.61	.54		
13. Obj Assem	.33	.74	.57	.34	.30	.16	.28	.31	.27	.51	.45	.68	
14. Dig Symb	.61	.78	.75	.52	.45	.48	.52	.54	.51	.49	.41	.74	.52
No Brain Damage	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**	**11**	**12**	**13**
1. VIQ													
2. PIQ	.68												
3. F-S IQ	.93	.91											
4. Inf	.83	.59	.78										
5. Comp	.76	.58	.74	.49									
6. Dig Span	.68	.45	.62	.44	.42								
7. Arith	.80	.51	.73	.59	.59	.33							
8. Simil	.81	.66	.80	.73	.63	.49	.46						
9. Vocab	.85	.63	.81	.84	.62	.60	.55	.78					
10. P Arr	.44	.70	.61	.32	.42	.34	.35	.39	.33				
11. P Comp	.66	.82	.81	.55	.65	.40	.44	.72	.61	.52			
12. Bl Design	.58	.81	.75	.42	.58	.37	.47	.59	.49	.50	.76		
13. Obj Assem	.14	.58	.38	.66	.20	.83	.14	.23	.14	.25	.45	.52	
14. Dig Symb	.74	.76	.81	.59	.60	.49	.56	.71	.70	.53	.65	.63	.26

TABLE 2.—INTERCORRELATIONS BETWEEN HALSTEAD'S NEUROPSYCHOLOGICAL MEASURES

Brain Damage	1	2	3	4	5	6	7	8	9	10
1. Imp Index										
2. Category	.71									
3. CFF	−.37	−.21								
4. CFF-Dev	−.21	−.08	.13							
5. TPT—Time	.42	.16	−.08	−.05						
6. TPT—Mem	−.27	−.04	−.18	.07	−.42					
7. TPT—Local	−.42	−.23	.10	.10	−.41	.58				
8. Rhythm	.49	.27	−.03	.18	.11	−.12	−.16			
9. Sp Perception	.58	.56	−.10	.26	.18	.02	−.11	.37		
10. Tapping	−.41	−.23	.13	.06	−.40	.21	.13	−.11	−.23	
11. Time Sense-Mem	.31	.13	−.16	−.16	−.08	−.03	−.08	.08	.04	−.03
No Brain Damage	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
1. Imp Index										
2. Category	.50									
3. CFF	−.12	.00								
4. CFF-Dev	−.17	−.01	.00							
5. TPT—Time	.50	.36	−.11	−.10						
6. TPT—Mem	−.51	−.29	−.05	−.20	−.32					
7. TPT—Local	−.61	−.40	−.02	−.13	−.26	.56				
8. Rhythm	.62	.27	.19	.17	.28	−.42	−.33			
9. Sp Perception	.51	.40	.00	.25	.29	−.40	−.23	.50		
10. Tapping	−.42	−.25	−.08	.17	−.18	.26	.04	−.33	−.32	
11. Time Sense—Mem	.23	−.07	−.16	−.12	.19	−.05	.01	.13	.00	−.24

damaged group and 23 of 55 coefficients in the group without brain damage. These matrices indicate that the interrelationships between Halstead's tests are much lower than for the W-B variables. Comparison of the 55 pairs of coefficients for the two groups indicates that in only 1 instance was the .05 level of confidence exceeded. In 55 comparisons this could easily be accounted for on a chance basis, indicating again that brain damage in general does not cause any substantial change in the interrelationships of various performances. Halstead's (5) matrix omits 3 measures (Impairment Index, Seashore Rhythm Test, and Critical Flicker Frequency-Deviation). His median coefficient was .27 as compared with .21 (no brain damage) and .14 (brain damage) when omitting these scales from the matrices in table 2.

Table 3 presents intercorrelations between Halstead's measures and the Wechsler variables for both groups. The median coefficients are .28 and .33 for the groups with and without brain damage. Eighty of 154 coefficients were significant beyond the .05 level in the brain-damaged group and 95 of 154 for the group without brain damage. Intergroup comparisons of the 154 pairs of coefficients indicated that only 4 were significantly different beyond the .05 level. Again the evidence indicates that relationships between performances are not differentially affected in a group of patients with heterogeneous brain damage as compared with a group having no brain damage.

The Impairment Index is probably the best indicator of biological intelligence among Halstead's measures, and the verbal, performance, and full-scale I.Q.'s the best indicators of psychometric intelligence among Wechsler's tests. The correlations between these measures are approximately the same for the groups with and without brain damage, and are of a magnitude of .55 to .64. These coefficients indicate a substantial relationship between these concepts of intelligence. While clearly not measures of exactly the same abilities, it appears that more than 25 per cent of the variance in one measure can be accounted for by the results obtained with the other.

TABLE 3.—INTERCORRELATIONS BETWEEN WECHSLER-BELLEVUE VARIABLES AND HALSTEAD'S MEASURES

Brain Damage	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)
(A)	−.55	−.55	−.58	−.55	−.32	−.47	−.50	−.39	−.44	−.38	−.41	−.54	−.41	−.41
(B)	−.58	−.64	−.65	−.55	−.52	−.45	−.50	−.43	−.45	−.53	−.55	−.65	−.53	−.53
(C)	.13	−.03	.00	.04	.04	−.07	.17	−.04	−.05	.09	.13	.07	.03	−.17
(D)	−.13	−.05	−.10	.09	−.02	−.25	−.10	−.28	−.08	−.13	.05	−.21	−.04	−.20
(E)	−.36	−.46	−.44	−.31	−.18	−.27	−.33	−.32	−.19	−.28	−.15	−.48	−.40	−.48
(F)	.08	.20	.17	.07	−.01	.08	.06	.06	.06	.16	−.03	.23	.18	.26
(G)	.19	.33	.29	.20	.02	.20	.28	.08	.10	.31	.07	.42	.24	.37
(H)	−.40	−.32	−.39	−.42	−.24	−.47	−.30	−.27	−.34	−.37	−.14	−.41	−.25	−.47
(I)	−.69	−.63	−.71	−.58	−.46	−.64	−.62	−.59	−.56	−.52	−.55	−.57	−.46	−.57
(J)	.31	.40	.38	.30	.20	.26	.23	.34	.20	.14	.37	.38	.27	.34
(K)	.09	−.10	.00	.05	.14	.11	.06	.06	−.02	.06	−.10	−.05	−.02	−.08
No Brain Damage	**(1)**	**(2)**	**(3)**	**(4)**	**(5)**	**(6)**	**(7)**	**(8)**	**(9)**	**(10)**	**(11)**	**(12)**	**(13)**	**(14)**
(A)	−.58	−.59	−.64	−.44	−.36	−.48	−.50	−.49	−.54	−.37	−.54	−.62	−.31	−.61
(B)	−.65	−.67	−.72	−.54	−.57	−.44	−.57	−.47	−.51	−.55	−.61	−.52	−.31	−.66
(C)	−.16	−.04	−.12	−.22	−.11	−.18	−.01	.00	−.24	.10	.01	.12	.19	−.09
(D)	−.28	.03	−.07	−.05	−.26	−.17	−.04	−.43	−.18	−.01	−.07	−.04	.10	−.09
(E)	−.18	−.49	−.36	−.12	−.15	−.10	−.19	−.20	−.20	−.44	−.33	−.57	−.34	−.38
(F)	.45	.42	.48	.30	.31	.46	.34	.28	.48	.25	.38	.33	.12	.32
(G)	.33	.37	.39	.23	.19	.35	.36	.18	.34	.31	.42	.41	.26	.32
(H)	−.53	−.36	−.50	−.40	−.33	−.52	−.30	−.50	−.55	−.22	−.42	−.31	.03	−.49
(I)	−.68	−.63	−.72	−.56	−.52	−.58	−.45	−.67	−.60	−.40	−.63	−.57	−.28	−.61
(J)	.45	.33	.42	.25	.50	.26	.29	.49	.38	.14	.37	.38	.07	.38
(K)	−.03	.03	−.01	.04	−.09	−.02	−.09	.01	.11	−.14	.09	−.10	.01	.03

Legend: (1) VIQ, (2) PIQ, (3) F-S IQ, (4) Inf, (5) Comp, (6) Dig Span, (7) Arith, (8) Simil, (9) Vocab, (10) P Arr, (11) P Comp, (12) Bl Des, (13) Obj Assem, (14) Dig Symb; (A) Imp Index, (B) Category, (C) CFF, (D) CFF-Dev, (E) TPT—Time, (F) TPT—Memory, (G) TPT—Localization, (H) Seashore Rhythm, (I) Speech Perception, (J) Finger Oscillation, (K) Time-Sense-Memory.

DISCUSSION

Previously obtained results on these same groups provide a background against which the above findings may be evaluated. Mean scores on Halstead's measures differentiated the groups strikingly. Not one brain-damaged patient obtained a better Impairment Index than did his matched control although several pairs had equal scores on this variable (9). The Wechsler variables also gave statistically significant intergroup mean differences although there was somewhat more overlap in the scores obtained by the groups (12). On nearly all measures the brain-damaged group consistently obtained poorer scores than the group without brain damage in terms of the quantitatively scaled level of performance. Nevertheless, the present results indicate that the interrelationships of various performances do not differ significantly from one group to the other.

These results indicate the importance of quantitative scaling as a basis for evaluating the psychological effects of brain damage. Many clinicians depend primarily upon qualitative evaluations, and some experimenters have argued that qualitative methods only are appropriate for understanding the effects of brain damage (4). The argument for qualitative evaluation usually postulates changes in relationship based on defects in quality or kind as the essential psychological result of brain damage. It is possible to postulate, however, that the changes are ones of degree along quantitatively scaled single variables. The present results strongly support this latter postulate. Our results indicate that the interrelationships of abilities remain approximately the same even when differences in degree (quantitatively scaled levels of performance) may be extremely pronounced. This result may be generalized only to the type of groups used in the present study. The possibility exists that other results might be obtained in

studying groups with differentially localized focal lesions.

The present results seem perfectly compatible with Halstead's interpretation of the factorial structure of biological intelligence. His C factor was interpreted as indicating abilities similar to those measured by scales of psychometric intelligence. Our results, then, support a conclusion that biological intelligence and psychometric intelligence are not separate or antagonistic concepts. Rather the concept of biological intelligence includes psychometric intelligence particularly as represented in its C factor. The possibility also exists that certain aspects of psychometric intelligence may contribute to other factors, but this hypothesis needs further investigation.

Summary and Conclusions

Halstead's ten tests, upon which the Impairment Index is based, and the Wechsler-Bellevue Scale of Adult Intelligence (Form I) were administered to 50 patients with proved brain damage and 50 control subjects who had no evidence of past or present brain damage or disease. The subjects in the two groups were closely matched in pairs on the basis of color, sex, chronological age, and years of formal education. Coefficients of correlation were computed for both groups for all possible pairs of variables among the tests given.

The results indicate that the W-B variables are generally much more highly intercorrelated than are Halstead's measures. Fairly substantial correlations were found between certain of Halstead's tests and the W-B variables. Significant differences between coefficients for the groups with and without brain damage occurred so infrequently that they can easily be accounted for on the basis of chance. Two conclusions are supported by the findings. 1) The concepts of biological intelligence (represented by Halstead's tests) and psychometric intelligence (represented by the W-B Scale) are not mutually exclusive. Our results are compatible with Halstead's original interpretation that the Central Integrative Field (memory) factor of biological intelligence is closely related to psychological tests of the I.Q. type. The possibility also exists that W-B variables might load significantly on other factors of biological intelligence. 2) While previous results have shown very substantial and highly significant differences in quantitatively scaled level of performance on the same tests in these same groups, the present study of possible intergroup differences in interrelationships of test scores fails to show differences in the groups with and without brain damage. The significance of this finding with respect to the role of possible qualitative as compared with quantitative changes resulting from brain damage is discussed.

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BIBLIOGRAPHY

1. Balinsky, B.: An analysis of the mental factors of various age groups from nine to sixty. *Genet. Psychol. Monogr., 23:* 191, 1941.
2. Birren, J. E.: A factorial analysis of the Wechsler-Bellevue Scale given to an elderly population. The Psychometric Lab., Univ. of Chicago, No. 73, 1951.
3. Cohen, J.: Factors underlying Wechsler-Bellevue performance of three neuropsychiatric groups. *J. Abnorm. & Social Psychol., 47:* 359, 1952.
4. Goldstein, K., and Scheerer, M.: Abstract and concrete behavior: An experimental study with special mental tests. Psychol. Monogr., No. 239, 1941.
5. Halstead, W. C.: *Brain and Intelligence: A Quantitative Study of the Frontal Lobes.* Chicago: Univ. of Chicago Press, 1947.
6. Halstead, W. C. Specialization of behavioral functions and the frontal lobes. The frontal lobes. *A. Res. Nerv. & Ment. Dis.*, 27–59, 1947.
7. Halstead, W. C. Biological intelligence. *J. Personality, 20:* 118, 1951.
8. McNemar, Q.: *Psychological Statistics.* New York: John Wiley and Sons, Inc., 1949.
9. Reitan, R. M.: Investigation of the validity of Halstead's measures of biological intelligence. *Arch. Neurol. & Psychiat., 73:* 28, 1955.
10. Reitan, R. M.: Affective disturbances in brain-damaged patients: Measurements with the Minnesota Multiphasic Personality Inventory. *Arch. Neurol. & Psychiat., 73:* 530, 1955.
11. Reitan, R. M.: Validity of Rorschach Test as measure of psychological effects of brain damage. *Arch. Neurol. & Psychiat., 73:* 445, 1955.
12. Reitan, R. M.: The effect of brain damage on a standard measure of intelligence. To be published.

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STUDIES ON MESCALINE

V. Electroencephalographic Evidence for the Antagonism between Mescaline and Chlorpromazine Hydrochloride[1]

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The observations reported in this paper were made during an extended study of the general psychophysiological effects of mescaline sulfate and chlorpromazine hydrochloride.[4] It was the purpose of this work to investigate electroencephalographically the physiological responses of the mescaline induced state. This data could help to contribute to an understanding of the clinical improvement seen in psychiatric patients following chlorpromazine hydrochloride administration.

The intravenous injection of 0.5 grams of mescaline sulfate produces a clinical state demonstrated by marked tension, anxiety with changes in sensory, motor, perceptual and thinking activity. The electroencephalogram reflects these changes most frequently as a decrease, disappearance, or rarely an increase in alpha waves (3). The slow waves and paroxysmal burst activity following electroshock therapy of schizophrenic patients are also suppressed by the intravenous injection of mescaline sulfate (12).

Intravenous mescaline produces apathy, lethargy, drowsiness or sleep in patients with proven convulsive disorder. The brain wave patterns consisting of dysrhythmia, paroxysmal as well as slow and spike wave discharges decrease or disappear for various periods of time. (4). These investigations (3, 4, 5, 6, 12) have led us to suggest that mescaline acts in the diencephalic centers, possibly upon the nuclear masses in the walls of the third ventricle.

Because of the central action of chlorpromazine hydrochloride and its depressant effect on vasomotor tone and reflex activity and the direct suppressing effect upon the reticular substance (2), we utilized the opportunity of testing its effects on the various components of the mescaline induced state in schizophrenic and epileptic patients.

The effects of chlorpromazine hydrochloride on the electroencephalogram in normal humans and animals have been reported elsewhere (14, 15). Bente and Itil (1) studied the effects of chlorpromazine hydrochloride on the electroencephalograms of epileptic patients showing paroxysmal and dysrhythmic patterns. They found the drug "manifests itself in an activation and provocation of paroxysmal dysrhythmias."

Materials and Methods

(a) Twenty-one male and female epileptic patients with clinical histories and electroencephalographic evidence of convulsive disorders with concomitant psychiatric disorders were studied. All patients were under treatment with individually determined doses of various anti-convulsant agents including dilantin, phenobarbital, hibicon, diamox and luminal. Three control electroencephalograms were done. At the end of the fourth recording each patient received a single intramuscular injection of 50 mgm. of chlorpromazine hydrochloride. The electroencephalogram was recorded for 43 to 109 min. following injection: the duration depending upon the length of time required for the tracing to return to the pre-test patterns. Twenty male and female epilelptic patients served as controls and received a single intramuscular injection of 2 cc. of physiological saline under identical conditions.

(b) Twenty-five male and female non-epileptic patients with various psychiatric diagnoses, mostly schizophrenia, were studied after the intravenous injection of 0.5 grams of mescaline sulfate followed 1–1½ hours later

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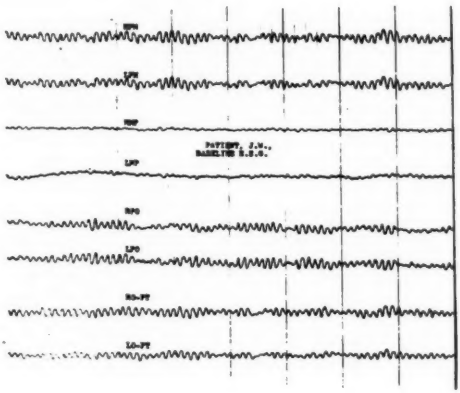
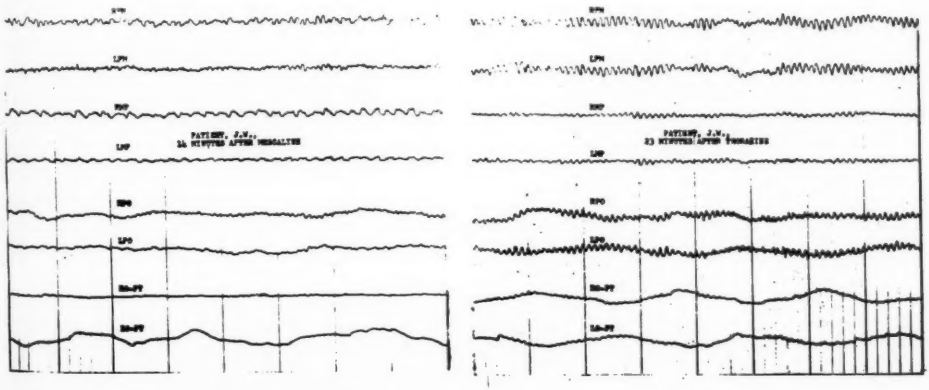
[4] Supplies of chlorpromazine and related pharmacological data were supplied by Mr. W. E. Kirsch of the Smith, Kline & French Laboratories, Philadelphia, Pennsylvania.

a b

FIG. 1a, 1). Electroencephalographic response demonstrating increased slow wave forms in epileptic patient, M. R., after Thorazine.



a



b c

FIG. 2a, 2b, 2c. Electroencephalographic response demonstrating the alterations of the pattern following mescaline with restoration of the baseline pattern following Thorazine.

by the intramuscular injection of 50 mgm. of chlorpromazine hydrochloride Electroencephalographic recordings were made in a similar manner as the epileptic patients. Ten patients served as controls with saline being substituted for mescaline; in 10 patients mescaline was injected and saline substituted for chlorpromazine hydrochloride; and in 10 patients saline was substituted for both mescaline sulfate and chlorpromazine hydrochloride.

Results

(a) *Figure 1.* In all 21 epileptic patients studied the intramuscular injection of chlorpromazine hydrochloride served to increase the abnormal brain wave potentials and provoked the accentuation of the paroxysmal and dysrhythmic activity.

The frequencies of the slow wave discharges decreased, and their amplitudes increased. There was an increase in the presence of spike wave discharges. This accentuated activity persisted for 50–90 min. following the injection. One patient suffered a grand mal seizure 12 minutes after the injection of chlorpromazine hydrochloride. Another patient complained of severe headache lasting 2½ hours. Sixteen of the 21 patients demonstrated a decrease or disappearance of muscle artifact and eye twitching as indicated in the electroencephalogram.

(b) *Figure 2.* Within two minutes following the injection of chlorpromazine hydrochloride, changes became evident in the electroencephalograms of the second group of 25 schizoprehenic patients. There was a reappearance of alpha activity with a return to the premescaline patterns in 17 of the 25 patients. Six patients who showed little change in the control patterns following mescaline showed an improvement in stability of the rhythm and an increase in amplitude following chlorpromazine hydrochloride which exceeded the pre-test patterns. Two patients could not be examined electroencephalographically. Clinically all signs of the mescaline induced state disappeared within approximately one hour after the injection of chlorpromazine hydrochloride. The changes in the electroencephalogram reflected this reaction.

Discussion

Our recent clinical observations on the central action of mescaline have led us to infer that the electroencephalographic patterns are a result of physiological changes occurring in the region of the diencephalon; possibly upon the nuclear masses in the walls of the third ventricle (12). The hypothesis has been tentatively confirmed by Elkes in experimental work in animals (7). The action of chlorpromazine on the central nervous system has been studied extensively by Hiebel *et al.* (10). It was the general impression that chlorpromazine hydrochloride:

1) Served to depress the responses of the reticular substances to sensory and nociceptor excitations producing a decrease in the excitability of the reticular mass.

2) Served to suppress the activating effects of adrenaline, in part, by diminishing central sensitivity and in part by reducing the peripheral circulatory response to adrenaline, and

3) Served to create favorable conditions for the increased central inhibition of the reticular mass by the carotid sinus afferent impulses.

The contributions made by Hess, Magoun and Moruzzi (8, 9, 11, 13) to our present understanding of the physiology of the reticular masses have served to help us hypothecate the antagonism between mescaline sulfate and chlorpromazine hydrochloride as indicated by the electroencephalogram. It has been shown that the maintenance of the waking state and the cortical electric activity that accompanies it depends on the continuous activity of the entire reticular substance occupying the central region of the brain stem, from the bulb to the posterior part of the hypothalamus. A disturbance of the system particularly in the anterior portion, leaving intact the regular routes of sensitivity up to the cortex, causes a suppression of waking activity and produces a suppression of the rhythmic patterns to that of one resembling sleep.

It would appear that the function of the reticular substance is that of an inhibitory mechanism in epileptic patients causing a control of excessive paroxysmal and dysrhythmic activity. With the introduction of chlor-

promazine hydrochloride there is a depression and a decrease in the excitability of the reticular mass thereby accounting for the activation of the abnormal pattern in the epileptic tracings.

With the utilization of mescaline sulfate causing motor, sensory, perceptual and thinking disorders and a decrease from the alpha patterns of the electroencephalogram, it is suggested that mescaline sulfate produced an excitation of some portion of the reticular substance which is restored following the injection of chlorpromazine hydrochloride. One might assume that a delicate balance exists which can be physiologically distorted utilizing pharmacological excitants such as mescaline and depressants such as chlorpromazine hydrochloride.

Summary and Conclusions

1. In a group of 21 epileptics the injection of chlorpromazine hydrochloride served to potentiate the abnormality of the brain-wave patterns.

2. Chlorpromazine hydrochloride in 25 schizophrenic patients serves to antagonize the clinical and electroencephalographic effects of mescaline sulfate.

3. Evidence is submitted to suggest the hypothesis that chlorpromazine hydrochloride by its depressant effects on the reticular masses serves to produce an accentuation of paroxysmal and dysrhythmic activity by the removal of the inhibitory activity of the reticular substance.

BIBLIOGRAPHY

1. Bente, D., and Itil, T.: Action of the phenothiazine derivative Megaphen on the human electroencephalogram. *Arzneimittel-Forschung, 4:* 418–423, 1954.
2. Dasgupta, S. R., and Werner, G.: Inhibition of hypothalamic medullary and reflex vasomotor responses by chlorpromazine. *Brit. J. Pharm. & Chemo., 9:* 389–439, 1954.
3. Denber, H. C. B., Merlis, S., and Hunter, W.: Studies on mescaline. I. Its action on the clinical and brain wave patterns of schizophrenic patients before and after electroconvulsive treatment. *Psychiat. Quart., 29:* 421–429, 1955.
4. Denber, H. C. B.: Studies on mescaline. III. Its action on the clinical and brain wave patterns of epileptic patients. *Psychiat. Quart., 29:* 433–438, 1955.
5. Denber, H. C. B., and Merlis, S.: Antagonism between mescaline and chlorpromazine. Presented at a combined meeting of the American Psychiatric Association and the A.A.A.S., December 30, 1954, Berkeley, California (to be published).
6. Denber, H. C. B., and Merlis, S.: A note on some therapeutic implications of the mescaline-induced state. *Psychiat. Quart., 28:* 635–640, 1954.
7. Elkes, J.: Personal communication.
8. Hess, W. R.: Das Zwischenhirn. Syndrome, Lokalisationen, Funktionen. Benno Schwabe & Co. Verlag. Basel, 1949, 187 pp., 2nd ed., 1954. (Summarized in English in: Gloor, P.: Autonomic functions of the diencephalon. A summary of the experimental work of Prof. W. R. Hess. *Arch. Neurol. & Psychiat., 71:* 773–790, 1954.)
9. Hess, W. R.: *Diencephalon. Autonomic and Extrapyramidal Functions.* New York: Grune and Stratton, 79 pp., 1954.
10. Hiebel, G., Bonvallet, M., and Dell, P.: Action de la Chlorpromazine ('Largactil', 45 60 WP) au Niveau du Systeme Nerveux Central. *Semaine d. hop. Paris, 30*(37): 2346, 1954.
11. Magoun, H. W.: Caudal and cephalic influences of the brain stem reticular formation. *Physiol. Rev., 30:* 460–474, 1950.
12. Merlis, S., and Hunter, W.: Studies on mescaline. II. Electroencephalographic response following electroconvulsant therapy in schizophrenic patients. *Psychiat. Quart., 29:* 430–432, 1955.
13. Moruzzi, G., and Magoun, H. W.: Brain stem reticular formation and activities of the EEG. *EEG Clin. Neurophysiol., 1:* 455–473, 1949.
14. Shagass, C.: Effect of intravenous chlorpromazine on the electroencephalogram. *EEG Clin. Neurophysiol., 7:* 306–308, 1955.
15. Terzian, H.: Electroencephalographic Investigation of the Central Action of Largactil (R.P. 4560) Ressegna di Neurologia Vegetativa. No. 4–5, 211–215, 1952. (Translated from the Italian by T. E. R. Singer, New York City.)

GOALS OF SHORT-TERM GROUP PSYCHOTHERAPY[1]

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A fundamental axiom of any psychotherapeutic procedure is the achievement of the maximum psychotherapeutic effect which a technique allows. The values, expectations, and accomplishments of a method are determined by its limitations. The results that are obtained in group psychotherapy can be judged fairly only in terms of goals that can be realized. A basic requirement in the practice of group psychotherapy is the formulation of goals. It gives the group psychotherapist a purpose to what he is doing and what he can anticipate from the group psychotherapy process. The group psychotherapist can evaluate more effectively the positive and negative results achieved by the psychotherapeutic effort.

A review of the literature discloses that many patients have gained an increased satisfaction with life as the result of the group psychotherapy experience. Pratt (9) in 1907, later through Harris, 1939 (2), attributed improvement to sthenic suggestions, friendly relations, and freedom from physical symptoms. He became the father of the suppressive-inspirational methods. Moreno (8), the inexhaustible drive behind the dramic therapies, reactivated in 1911 the concept of the collective catharsis of Greek tragedy—a catharsis for both the actor and the audience. Lazell (6), the pioneer of group psychotherapy with psychotics, contributed the goal in 1921 of the reduction of the fear of the therapist as an individual. Behind the valiant web of obfuscation spun by Burrow (1) in 1927 is the resolution of the sense of isolation in conflicts through the analysis of immediate mood changes. Marsh (7) published his zealous attempts to return the psychotic back to sanity by means of revivalist methods in 1931. His chief improvement criterion was the conversion of the patient's day-dreaming fantasy life into a sharper reality awareness. In 1938, Schilder (10), standard-bearer of the reconstructive methods, set forth his views that patients had to learn many things in order to gain insight into the components of their illnesses. Slavson, 1943 (11), the most prolific writer in the history of group psychotherapy, has outlined therapeutic changes in terms of dynamic factors. Subsequent writers in group psychotherapy have used the psychotherapeutic factors described by the above authors in one way or another, either singly or combined.

Do most practitioners of group psychotherapy *operate* with a clear definition of goals towards which group psychotherapy is geared? Are the real needs of patients often satisfied in a floating manner without a guiding target? Perhaps by experimenting further on previously suggested goals, there may emerge a more crystal delineation of psychotherapeutic goals as they relate specifically to short-term dynamically-oriented group psychotherapy.

Group psychotherapy is defined as the development of verbal and emotional interactions and part-identifications in an initial collection of unrelated malfunctioning individuals, led by a qualified psychotherapist, purposely motivated toward the common goal of the alleviation of reality problems on a conscious level (3). It is hypothesized that the group organizes gradually into a functioning whole by means of various levels of interlocking relationships and identifications.

PROCEDURE

A pilot study[2] was conducted, based on patients' written comments at the conclusion of four months of one and a half hour weekly sessions, and cataloged in accordance with apparent indices of improvement. These groups consisted of white, male, adult psychoneurotics, ages 19–44, and were conducted in dynamically oriented (4) tradition. On the basis of clinical experience and by definition, the fol-

[1] Presented at a teaching session on group psychotherapy in the State Hospital at Raleigh, North Carolina, on March 28, 1956.

[2] Originally formulated with J. Weinberger, M.D., in 1950, with subsequent modifications and developments by the author.

lowing typology was tentatively assigned an ascending order of importance, ostensibly portraying various levels of group participation in a chain of increasing relationships and part-identifications: (1) information, (2) social-participation, (3) loss of isolation, (4) loss of fear of personal expression: (a) speech, (b) and/or feelings, (5) increased confidence (reassurance), (6) symptomatic relief: (a) emotional, (b) and/or physical, (7) insight: (a) intellectual understanding of the self, (b) increased objectivity.

Definitions. These seven psychotherapeutic goals were defined in the following way:

(1) Information is the acquisition of new knowledge about emotional health. It is a level of isolated factual gain without advancing any meaningful encroachment upon the individual's understanding. Examples: "I learned a few things I did not know before." "Just gained some valuable knowledge—nothing more."

(2) Social-participation is the intellectual participation by an individual without becoming part of the group's emotional relationship to a significant degree. It is the initiating level to possible further interaction. Examples: "They were a fine bunch of fellows." "I am more sociable to people without resorting to sarcasm."

(3) Loss of Isolation is the presence of a common factor conveying emotional feeling in the individual with some members of the group. It heralds the return of the patient to a competitive social orbit. Examples: "I am not alone; there are others just like me."

(4) Loss of Fear of Personal Expression is defined as the voicing of conscious feelings without the fear of leader or group. This category includes two aspects:

(a) Loss of Speech Inhibition indicating, simply, the increased ability to talk. Examples: "I talk more openly and unguardedly and not as carefully as I used to." "The doctor was O.K. He let us talk about anything we wanted."

(b) Release of Feelings (abreaction) referring to the increased ability to talk with affect. Examples: "Expressing things out in the open are not as magnified as when bottled up inside." "Blowing off helped."

(5) Increased confidence (reassurance) is a loss of feelings of inadequacy either partially or totally. Examples: "Confidence has improved. I have a little more security."

(6) Symptomatic Relief is the patient's statement that he feels better emotionally or physically. It may occur with or without insight. Examples: "My crazy thoughts do not come as often." "The rash I had on my legs is gone."

(7) Insight, may be represented by two levels:

(a) Intellectual understanding of the self: This is a reduction (through explanations) of initial and continued resistance towards thinking that symptoms may possibly be emotionally occasioned. Example: "I now realize how certain thoughts can affect me physically."

(b) Increased Objectivity: This is a dissociation of the self from emotional attitudes. It is seeing the self in two pieces. The dissociation created by the conflict is driven into the open. Insight, on this second level, refers to the reinstatement of feelings and thoughts that were at one time *conscious*, but subsequently repressed. Example: "I have been able to face myself."

Reliability. How well could these goals be identified a second time from the written protocols? Provided with definitions of these criterion variables, could others learn and identify them? For this purpose, a fourfold point correlation was obtained between the two ratings by the same judges of a random sample of 25 cases. The two ratings were independent, but the judges worked together both times. The results are illustrated in table 1.

Table 1 shows that loss of speech inhibition is more difficult to identify, in the use of the

TABLE 1.—RELIABILITY OF IDENTIFYING GOALS

Goals	Ratios	
1. Information	1.0	
2. Social-participation	1.0	
3. Loss of isolation	1.0	
4. Loss of fear of personal expression	.78	
a. Speech		.51
b. Feelings (Abreaction)		.84
5. Increased confidence	.76	
a. Reassurance		.68
b. Symptomatic relief		.56
6. Insight	.76	
a. Intellectual understanding of self		.68
b. Increased objectivity		.51

written protocol alone, than increased expression of feelings, and was therefore tentatively assigned for statistical purposes to the broader category of the loss of fear of personal expression. Emotional symptomatic relief could not be clearly differentiated in the comments of these patients from increased confidence, and increased objectivity could not clearly be differentiated from intellectual understanding of the self. There were more scorable statements for identifying increased confidence and intellectual understanding of the self than there were for identifying emotional symptomatic relief and increased objectivity. For the purpose of increasing appropriate "reliability", symptomatic relief was tentatively subsumed under increased confidence and increased objectivity under insight. The categories of speech inhibition, symptomatic relief, and increased objectivity are really different from the categories with which they were merged, but the verbalizations in the above sample were too scanty to permit the differentiations required for a post-constructed classification of manifest psychotherapeutic gains.

In order to obviate the difficulties originally experienced in setting up a pilot classificatory system, a list of possible effects and benefits of group endeavors was prepared in the form of a more sharply discriminating series of statements and presented to the new groups of patients at the completion of group psychotherapy. These patients were requested to check as true or false, and to comment upon each statement sampling "the following effects that have occurred to you as the result of group treatment." These benefits were phrased as follows:

1. I have learned a few things about this subject I did not know before.
2. I can mingle with people more now.
3. I have lost some of my feelings of being different from people.
4. I can talk more readily.
5. I let go bottled-up feelings.
6. My confidence has improved; I feel more secure.
7. I feel better physically.
8. My wild feelings do not come as often or as strong as before.
9. I have come to realize how certain worries can affect me.
10. The group helped me to face, directly or indirectly but more realistically, certain important facts in my own experiences that I would have otherwise missed or avoided.

Results

The protocols of 20 white male patients who were discharged as improved were compared with 20 white male patients who were discharged as unimproved. Both the improved and unimproved patients were selected at random out of 18 groups led by seven different group psychotherapists with a dynamic orientation. The terminal impression as to who was improved or unimproved was determined by an interviewer other than the group psychotherapists. The patient population had a mean chronological age of 28 years and a mean educational level of 12 years. Marital status and major categories of occupation were similarly distributed. The majority of patients was Catholic. Patients met once weekly for one and a half hour sessions. All non-psychotic patients who were referred to group psychotherapy were automatically accepted. The reasons for referral to group psychotherapy were varied: orientation to psychotherapy (45%), support (27%), witnessing that other people had difficulties (14%), socializing outlet (11%), and stimulation of free association for individual psychotherapy (1%).

Table 2 presents a comparison between the improved and the unimproved groups in goals obtained in relationship to discharge status.

TABLE 2.—A Chi Square Test of Significance Between Patients Discharged as Improved and Unimproved with Relation to Obtained Goals

Goals	Chi Square Value	Level of Confidence
1. Information	1.66	.20
2. Social-participation	3.12	.05–.10
3. Loss of isolation	8.32	.001–.01
4. Loss of fear of releasing	11.78	.001
a. Speech		
b. And/or feelings		
5. Increased confidence (reassurance)	12.10	.001
6. Symptomatic relief	5.58	.02–.01
a. Physical		
b. And/or emotional		
7. Insight	2.06	.20–.10
a. Intellectual understanding of self		
b. And/or increased objectivity		

Information, social-participation, and intellectual insight were not statistically discriminated between the improved and the unimproved groups. Loss of fear of releasing speech and/or feelings, loss of isolation, increased confidence, and symptomatic relief (physical and/or emotional) appeared to be goals capable of being realized in an improved group exposed to short-term dynamically oriented group psychotherapy.

Discussion

It is hypothesized that group psychotherapy is the evolution of a process of part-identifications which occur in a chain-like and interlocking fashion. The goals of group psychotherapy are attained at various levels *pari passu* with identifications.

Information and social-participation. A collection of illness *per se* does not constitute group psychotherapy. Treatment does not occur unless it is sought. Group psychotherapy must have a rational and scientific framework. Like all other organized groups, it must satisfy a demand for appropriate and pertinent information. Only pertinent material becomes incorporated in their understanding.

Some patients are too narcissistic to permit more than peripheral contact. They may timidly approach psychotherapists for reading material as their sole major bid for psychotherapy. These patients tolerate the presence of people as long as the acquaintance is casual, infrequent, formal, and impersonal. The group is not much more than a club meeting, "one night out." However, even these patients come for a special type of information, but with the express determination that they are not going to become emotionally involved. "I honestly believe I attended the group to hear what the conductor had to say." " I feel definitely detached from all members of the group." "I trained my mind not to let myself care enough for anyone or anything." "They were a very nice group, but I have no friends and desire none." "I cannot fulfill the obligations or take on the extra burdens of friendship." Some patients mingle with other individuals on a friendly level, but they are not a part of a cohesive unit. There is a social stimulus but no interstimulation. Other patients welcome the opportunity for social contacts. For example: "This combined pleasure with a job to be done." Still others lunch with each other, are in telephonic communication, or form the beginning of new friendships. Those members who do not advance beyond the goals of information and social-participation fail to meet the identification standard required for the effective group treatment of neurosis.

Clubs do offer information, consistent with their charter, and the opportunity to make new friends. Unlike group psychotherapy, the club places no pressure upon the individual to step out of the orbit of personal isolation. The above scale of goals was administered to members of a club following four months of its existence. More members obtained the goals of information and social-participation who joined a psychotherapy group as compared with the club (chi square 7.22, p.01–.001). Variations in "levels of confidence" may be anticipated when group psychotherapy is contrasted with other types of group organizations. However, it is unlikely that any additional goals will be achieved. The active members in a club are active at the outset and continue as such, while the passive members remain frozen in their impasse.

Loss of isolation. The attainment of this goal ties the individual to a group on his own emotional level, and on the basis of this similarity, the members of the group develop an emotional relationship to each other. They are brought face to face with what they have in common with other members. There is a badge of membership. An overevaluation of the self occurs, e.g., "I am not so bad off." There is a reorientation of one's self in relation to a new experience. In the group, they no longer feel alone against the world. Their anxiety is lessened in the feeling that they are not alone. They belong even if it is a group of fellow sufferers. For example: "I look around and see that I am not the only one to come with troubles." Some asocial patients do not permit the conscious recognition of being alone. For example: "I never mind being alone." "I never felt important enough to be missed."

Loss of fear of personal expression. Most of the patients who experienced a decrease of speech inhibition also experienced a dramatic siphoning of feelings. Although "speech" and "feelings" were combined for statistical purposes, because one usually occurred with the

other, they will have to be calculated separately for other groups for precisely the opposite reason. For example, in a group conducted in a military hospital during peacetime, 14 of 16 corpmen checked the statement that they talked more readily, but only 2 out of 16 corpmen indicated that they were able to release bottled-up feelings!

There is an abeyance of the superego by identification. Loss of isolation in group psychotherapy with the neuroses precedes the loss of fear of personal expression: "When I saw others had similar problems, I was interested and talked." They are stimulated towards a release of these feelings on the initiative of other members. The decrease of isolation may be reinforced through interaction: "These feelings are not just mine. The group has decided that for me. Others have the feelings I do." A gathering of emotionally disturbed patients provides a "group climate" where they can express their special needs. "I've expressed some feelings that I've been shielding for years without finding the appropriate persons to thrash them out with." In the spontaneity of the group, they discover that other people have similar attitudes. Here they can admit their feelings of inferiority, weaknesses, and unacceptable impulses, whereas outside the group people "would think you were crazy." Here patients can relax, laugh, talk, and face their problems. Outside the group, isolation occurs immediately: they become tense, cannot smile or talk to people; they cannot adjust, they feel weak and tremble.

The sickest patients do not find this relaxation and reduction of guilt and isolation: "I never could express my feelings because it was never done in my family." "Although I enjoyed the discussions very much, I still feel I'm a misfit. People just do not take to me. I cannot understand why." "I was afraid to talk about something because I know I would receive the disapproval of the group."

The patients' progress from silent, passive participation to the freeing of speech and feelings in active participation is a monumental feat leading to the higher goal of increased identification, ego strength, reassurance.

Increased confidence (*reassurance*). Statistically, reassurance can be depended upon with an equal "level of confidence" as loss of fear of personal expression in short-term group psychotherapy. Both goals are intimately interwoven. Reassurance automatically occurs with loss of fear: "Talking in these sessions has given me more confidence in myself." More specifically, in group psychotherapy, loss of isolation plus loss of fear of personal expression (abeyance of the individual's superego) enhance the maturation and development of the goal of reassurance. Some of these factors may be stated as follows:

(1) The group provides figures who are sick and can speak positively as fellow living sufferers on an *equal* status. "I feel that trying to solve a problem in a group of people with the same problem gives me a feeling of strength and confidence." Members can express similar emotional needs in terms of their conflictual situations.

(2) The group provides figures who appear *permissive* to others. There is a more simplified, less threatening, less competitive culture. Competition is still present, but it is with sick siblings instead of assumed well siblings. Patients are allowed to express hostilities, guilt feelings, anxieties, fears, and yet be socially acceptable. "Everyone seemed to express his feelings freely with no regard for shame or personal information. This I feel made me say the things I wanted to with a little more security." Members of the group find they can express all these feelings and nothing happens. They are accepted and it is safe, and in that safety there is reassurance.

(3) The group provides figures who appear *stronger* to others. These figures are not afraid or inhibited in those areas in which others express fear and constraint. These figures serve as a model or example of emotional security and, in this way, a support. "I have found something to like and admire, sometimes wishing I had some of these qualities I was discovering in the group."

(4) The group provides figures who appear *sicker* to others. "I felt that I had as much if not more intelligence than the others and because of that I gained confidence." "No matter how tranquil a person appears, he may have problems bothering him which are akin to mine, if not worse." Patients acquire a deep and irrevocable reassurance that they are not

the weakest, the most disgusting, the most distorted representatives of society. Patients become reassured that although other patients appear to be sicker than they, these other patients are still ambulatory and serve as a sign of good health despite their label of ill health.

Symptomatic relief. As one proceeds from the more general and the more common to the specific and less frequent changes, the less likely is there apt to be psychotherapeutic success. For example, a number of patients report that they are generally improved, but substantially fewer patients report complete alleviation of specific symptoms. One is forced to assume that the physical expression of conflicts may be more difficult to yield to short-term group psychotherapy. Apparently, some patients are not aware of affective disturbance, its translation into physical terms; or the adjectival description in the statement of goals did not apply with sufficient concreteness to their particular condition.

Insight. A number of patients checked this goal automatically. In part, this is probably due to positive transference, but mostly to misunderstanding and distortion of area explored. Objective scoring of this goal showed that the first level of insight (intellectual understanding of the self, as defined above) was infrequently achieved, and the second level (increased objectivity) was a rare occurrence. To increase the "reliability" of such instruments, it is strongly recommended that each statement, in the hierarchy of goals, is carefully reviewed with the patient by an impartial observer. It cannot be assumed that the average patient has sufficient technical sophistication to be able to determine what specialists themselves are laboring to differentiate.

Certain attitudinal types of resistances were observed in comments to statements of goals by patients. The first level of insight was equated with an academic acquisition, e.g., information only. The second level of insight was interpreted by patients as loss of isolation ("Perhaps my case to another's point of view may have seemed hopeless"), as a detached, impersonal encounter ("Hearing everyone's views gives us all sides of the story"), or because insight on either basis had no recognizable meaning. While the first level of insight may be within the possible reach of some patients in short-term group psychotherapy, the second level of insight can only be realized in extensive, intensified approaches (5). Patients have a tenacious tendency to ignore reality correction, and modification in their mechanisms of displacement, projection and their modality or pole of libidinization.

A supplementary survey indicated that patients carry over into their daily living the benefits of group psychotherapy. Some members spend time the rest of the week following the group session in thinking ("Yes, I think about the sessions and review all that has happened until I go again to the next one." "I live day to day waiting for the group session."), in discussing the sessions with member of the family and friends, or acting upon what has taken place in the group ("I have put into practice many ideas formed at these sessions." "I have undertaken two projects that I had been reluctant to attempt for some time.").

Summary

An attempt is made to set realistic sights as to the goals that can be achieved in dynamically oriented short-term group psychotherapy with adults suffering from emotional disabilities. Progress is interpreted in the light of levels of interlocking relationships and identifications. A classification system was constructed for the purpose of cataloging indices of improvement in terms of a clinically assigned order of goals of ascending importance, and subjected to a reliability test. Goals realized by discharged improved patients were compared with those of discharged unimproved patients by means of a statistical test of significance. Loss of fear of releasing speech and/or feelings, loss of isolation, increased confidence (reassurance) and symptomatic relief (physical and/or emotional) were significantly related to improvement. A clinical discussion and documentation of this series of goals were presented.

BIBLIOGRAPHY

1. Burrow, T.: The group method of analysis. *Psychoanal. Rev., 14:* 268–280, 1927.
2. Harris, H. I.: Efficient psychotherapy for the large outpatient clinic. *New England J. Med., 221:* 1–15, 1939.

3. Kotkov, B.: Research. In *The Fields of Group Psychotherapy*. S. R. Slavson, Ed. New York: International Universities Press, Inc., 1956.
4. Kotkov, B.: Dynamic Techniques of Group Psychotherapy. *To be published.*
5. Kotkov, B.: Analytically oriented group psychotherapy of psychoneurotic adults. *Psychoanal. Rev., 40:* 333–350, 1953.
6. Lazell, E. W.: The group treatment of dementia praecox. *Psychoanal. Rev., 8:* 168–179, 1921.
7. Marsh, L. C.: Group treatment of the psychoses by the psychological equivalent of the revival. *Ment. Hygiene, 15:* 328–349, 1931.
8. Moreno, J. L.: Die Gottheit als Komoediant. Anzengruber Verlag, Vienna, 1911.
9. Pratt, J. H.: The class method of treating consumption in the homes of the poor. *J. A. M. A., 49.* 755–759, 1907.
10. Schilder, P.: *Psychotherapy*. New York: Norton, 1938.
11. Slavson, S. R.: *An Introduction to Group Psychotherapy*. New York: Commonwealth Fund, 1943.

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PROMAZINE HYDROCHLORIDE IN THE TREATMENT OF CHRONIC CATATONIC SCHIZOPHRENICS

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Promazine hydrochloride[1] (hereinafter referred to as promazine) is a phenothiazine compound which differs from chlorpromazine hydrochloride in that promazine does not have a chlorine atom substituted on the phenothiazine ring. Fazekas *et al.* (1, 2) have found promazine to be effective in the management of acute drug and alcohol induced syndromes and in calming acutely disturbed psychotics (manic depressives and agitated schizophrenics). Mitchell (5) confirmed the findings of Fazekas *et al.* in the management of acute alcohol induced syndromes.

Purpose of the present study was to make an initial assessment of the effectiveness of promazine in the treatment of chronic, regressed, catatonic schizophrenic patients.

METHOD

Previous treatment methods had been unsuccessful with the 30 patients in this study. Twenty-seven of the 30 had been treated with electric convulsive therapy and/or with insulin coma therapy, and three of these 27 had also undergone a prefrontal lobotomy. Age range was from 25 to 63; median age was 35. Years continuous hospitalization ranged from 1 to 20; median was 10.

Method of drug administration was primarily oral with only occasional intramuscular injections. Dosage was started at 50 mg., q.i.d., was stepped up by 50 mg. every three days until the fourth week when a dosage of 450 mg., q.i.d., was reached. At this time, there was a four-day break in the drug supply so that the dosage was reduced to 450 mg., b.i.d., for one day; to 50 mg., b.i.d., for one day; to nothing for two days; then back to 100 mg., t.i.d., for one day; to 100 mg., q.i.d., for one day; then to a maintenance dosage of 200 mg., t.i.d.

Behavioral evaluation was accomplished by three procedures: 1) Each patient was seen for a clinical psychiatric interview immediately prior to treatment, again 3 months after treatment began, and again 6 months after treatment began. At the 3-month and 6-month interviews, a clinical judgment was made as to degree of change from the patients' pre-treatment level of adjustment. This judgment of change was stated in terms of the usual clinical judgment scale, i.e., marked improvement (thinking, affect, and behavior approaching normality), moderate improvement (more comfortable, less of a management problem, no longer mute, etc.), slight improvement (noticeable improvement in any particular), and no change. 2) A standardized interview[2] was developed wherein 30 specified behaviors were observed for and then checked as to presence or absence. Each behavior was assigned a weight of one. Total score for this standardized interview could, then, range from zero to 30. Those behaviors were selected on the basis of their relative simplicity so that even minimal changes in behavior could be discriminated. This interview was conducted at the same intervals as the clinical psychiatric interview. Inter-rater reliability was high, rho = .96. 3) The Ward Observations section (items 41–62, excepting items 46, 49, 51, 54, 59, 61) of the Multidimensional Scale for Rating Psychiatric patients (4) was completed by the psychiatric aide at the same time intervals as the above procedures. A total scale points deviation from the normal pattern (possible scores zero to 40) was obtained from this scale. These procedures provided us with three independent evaluations of behavioral change which are referred to as the clinical psychiatric, standardized interview, and ward behavior, respectively.

RESULTS AND DISCUSSION

Table 1 shows the changes from pre-treatment to 3 months and from pre-treatment to 6 months as determined by the three pro-

[1] Supplied for this project by Wyeth Laboratories, Philadelphia, Pennsylvania (trademark "Sparine").

[2] Developed and administered by psychology trainees Amerigo Farina, David L. Arenberg, and Samuel L. Guskin.

TABLE 1.—CHANGES IN BEHAVIOR WHILE ON PROMAZINE MEDICATION

Clinical Psychiatric			Standardized Interview			Ward Behavior		
Change	3 mos.	6 mos.	Change	3 mos.	6 mos.	Change	3 mos.	6 mos.
Improvement								
Marked	0	0	+13 up	0	0	+18 up	0	0
Moderate	6	6	+8 to +12	3	3	+11 to +17	1	1
Slight	6	9	+3 to +7	10	16	+4 to +10	8	7
No change	18	14	−2 to +2	16	9	−3 to +3	19	20
			−3 to −7	1	1	−4 to −10	2	0
			−8 to −12	0	0	−11 to −17	0	1
N	30	29*		30	29*		30	29*
Mean change	—	—		2.13	3.52		1.53	2.24
t	—	—		3.13	5.42		2.11	2.82
P	—	—		<.01	<.001		<.05	<.01

* One patient lost by transfer for treatment of pulmonary tuberculosis.

cedures. Plus values represent gains, and minus values represent losses in total score. The changes in patient behavior are in the direction of improvement and are statistically significant. Practical significance (as contrasted with statistical significance) was also deemed an important yardstick for assessing the value of a treatment method, however, and by this yardstick there was little about which to become enthusiastic. The average gain was minimal. There were no dramatic recoveries or even striking improvements. Correlations among the three evaluation procedures were quite low (median r = .23), suggesting that they tapped minimal variations in different aspects of patient behavior. From these considerations of the data, it is doubtful if any practical therapeutic significance can be ascribed to promazine medication (alone and in the dosages here prescribed) with long-term catatonic schizophrenic patients. It was the clinical impression of the senior author and ward personnel that the patients may have become somewhat more relaxed during the treatment period, but increased relaxation alone in chronic catatonic patients is a difficult variable to assess.

A comment in regard to methodology may be appropriate in the discussion at this point. In this study, each subject served as his own control. Had we been able to conclude from our data that therapeutic gains of practical significance had accrued during the course of treatment, then a follow-up study with matched control subjects would have been indicated in order to parcel out the effects of such variables as patients' knowledge that they were receiving a new medication, increased attention from personnel, and experimenter bias. A recent study by Hall and Dunlap (3) presents a clear discussion of the hazards in making positive conclusions from drug studies without adequate safeguards of a controlled design. In our design, the limited number (local) of specifically designated chronic, regressed, catatonic schizophrenic patients were conserved in one experimental group of sufficient size so that what may have occurred with treatment was given ample opportunity to occur. Since we have concluded from the data that any practical therapeutic advantage of the medication (as prescribed and with chronically ill subjects) under study is doubtful, a controlled follow-up study is not indicated.

SIDE EFFECTS

Though it was not our primary purpose to study the physiological side effects of promazine, the usual precautions in administering a new drug preparation were adhered to, and the consequent observations are, accordingly, presented as a matter of record.

There was an average increase in weight of eleven pounds ($P < .01$) during the first three months of treatment and an additional average increase of five pounds ($P < .01$) during the second three months. The weight gain was more often regarded as a desirable rather than an undesirable side effect. With few exceptions, resting blood pressure, alkaline phosphatase, white blood count, and differential counts all

showed minimal changes during the study. Diastolic pressure showed a slight average drop from 75 mm. Hg to 71 mm. Hg during the first three months. Neutrophils showed a slight average increase from 59 per cent to 63 per cent during this interval. Alkaline phosphatase showed a slight average increase from 3.30 to 4.17 Bodansky units during this interval. From pre-treatment to six months there were no average trends of any importance in any of these variables (alkaline phosphatase was dropped from the routine laboratory test schedule after the first three months).

First toxic symptoms occurred with one patient fainting at the 200 mg. daily level and again at the 600 mg. daily level. At 800 mg. daily, another patient fainted. Upon reaching the 1,400 mg. daily level, two patients developed marked generalized tremor and fell to the floor. One of these patients was placed on a reduced maintenance dosage of 800 mg. daily and experienced no further difficulty.

The second of these two patients suffered a linear skull fracture from his fall. Promazine medication was discontinued, and he was given penicillin, vitamin C, and intravenous dextrose. On the sixth day after his fall his white blood count was 2,750, neutrophils 0, lymphocytes 99, monocytes 1. He had a 48-hour rise of temperature reaching a maximum of 105° (R). He received further treatment with penicillin and was given three blood transfusions. Neutrophils reappeared on the eighth day, and on the eleventh day the white blood count was 8,450 with a normal differential. The cause of this agranulocytosis was presumed to have been the administration of promazine, but the possibility of other factors in the etiology such as skull fracture or an intercurrent virus infection was considered. On the 31st day after skull fracture, the administration of promazine was cautiously resumed and built up to a dose of 600 mg. daily, which he has subsequently received for a period of five months without difficulty of any kind.

Upon reaching a dosage level of 1,800 mg. daily, four more patients developed marked tremor. Two of these fainted; one became hyperactive.

It was at the 1,800 mg. level that there was a 4-day break in the drug supply. Upon withdrawal, five patients showed transient vomiting. One patient showed repeated vomiting and was given 100 mg. promazine intramuscularly with relief. Two other patients showed marked agitation upon withdrawal. After the drug supply was renewed and medication was resumed, symptoms of vomiting and agitation were relieved.

During the last three months of the study and with a median maintenance dosage of 600 mg. daily, there has been no recurrence of any toxic symptoms. At no time have there been any such complications as jaundice, dermatitis (of either patients or personnel), edema, or parkinsonism. Treatment interval was from October to April so that the patients were not exposed to severe insolation. With the few intramuscular injections there was no local tissue reaction even in doses up to 250 mg. in 5 cc. of water.

Summary and Conclusions

A new phenothiazine derivative, promazine, was administered to a group of 30 chronic, regressed, catatonic schizophrenic patients (median years continuous hospitalization: ten) in an initial assessment of its therapeutic effectiveness with such patients. Dosages were stepped up to 1,800 mg. daily, then reduced to a maintenance dosage of 600 mg. daily. The treatment period extended over six months. Behavioral evaluation was accomplished by three independent procedures: Clinical psychiatric judgments, a standardized interview, and ward observations. The changes in patient behavior were in the direction of improvement and were statistically significant. The average gain was minimal, however, and there was no individual instance of marked improvement. It is our conclusion that little or no therapeutic efficacy of practical significance can be attributed to promazine medication (as administered in this study) with the chronic, regressed patients under study.

Side effects were of no consequence at dosage levels up to 1,400 mg. daily. At a level of 1,400 to 1,800 mg. marked tremor developed in six patients. Tremor was followed by fainting in four of these six. Reduction to a maintenance dosage relieved these symptoms. One instance of agranulocytosis developed which responded to withdrawal of promazine and appropriate treatment. With adequate

safeguards this patient was again placed on promazine with no ill effects. Through a break in the drug supply, the patients were two days on reduced dosage and two days with no medication. Six patients showed vomiting upon withdrawal which cleared upon resumption of treatment. Such complications as jaundice, dermatitis, edema, and parkinsonism have not been noted.

In view of the extreme chronicity and general lack of agitation of the patients under study and the low order of toxicity observed (relative to that reported in the use of chlorpromazine), it is suggested that promazine be tried with a less chronic and more disturbed group of schizophrenic patients.

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BIBLIOGRAPHY

1. Fazekas, J. F., Sullivan, P. D., and Schultz, J. D.: Use of dimethylamino-n-propylphenothiazine in the management of patients with acute mental disturbances. *Med. Ann. D. C., 25:* 67–69, 1956.
2. Fazekas, J. F., Schultz, J. D., Sullivan, P. D., and Shea, J. G.: Management of acutely disturbed patients with promazine (Sparine). *J.A.M.A. 161:* 46–49, 1956.
3. Hall, R. A., and Dunlap, D. J.: A study of chlorpromazine: Methodology and results with chronic semi-disturbed schizophrenics. *J. Nerv. & Ment. Dis., 122:* 301–314, 1955.
4. Lorr, M.: Multidimensional scale for rating psychiatric patients: Hospital form. *V. A. Technical Bulletin (TB 10-507)*, 1953.
5. Mitchell, E. H.: Treatment of acute alcoholism with promazine (Sparine). *J.A.M.A., 161:* 44–45, 1956.

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FURTHER STUDIES OF THE PSYCHOLOGICAL EFFECTS OF FRENQUEL AND A CRITICAL REVIEW OF PREVIOUS REPORTS[1]

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I. Introduction

Interest in Frenquel[3] was considerably stimulated by Fabing's report (2) that this drug effectively "blocked" the psychological effects of LSD-25 and mescaline. Fabing as well as others (3, 5, 6) has since carried out clinical trials with Frenquel on a variety of conditions in which hallucinations or delusional thinking were conspicuous features. These have included acute and chronic schizophrenia, delirium tremens, post-operative confusional states and senile dissociation syndromes. It was apparently assumed that if Frenquel blocked the effects of hallucinogenic drugs, it might be beneficial in other forms of mental disturbance.

The relationship, if any, of the psychological phenomena induced by hallucinogenic drugs to disorders such as schizophrenia is a controversial question which will not be considered at length in this paper. Serious objections have been raised to use of the term "model psychosis" to characterize intoxication with LSD-25 or mescaline. It has been felt that the term itself might be misleading and in any event was not justified by available evidence. However, in the reported studies of the "antihallucinatory" effects of Frenquel, it has been tacitly assumed that hallucinations, for example, produced by LSD-25 or occurring in delirium tremens are the same as, or at least are comparable to, the hallucinatory phenommena of schizophrenia or other functional psychoses and might be similarly modified by the drug.

Even at a descriptive level, there is reason to be doubtful about this assumption. In toxic delirium, visual hallucinations are detailed, vivid, often colorful, kaleidoscopic, and variously farcical or frightening in content. These characteristics have, in fact, been used clinically to identify hallucinations of toxic origin. Visual hallucinations in schizophrenics, if one excludes cases complicated by metabolic disturbances such as severe dehydration, infection, etc., are usually vaguely described in terms of detail or color, stereotyped, symbolic, and frequently meaningful as projected fantasies. Auditory hallucinations are observed far more frequently in schizophrenia than visual hallucinations. The latter are of course common in toxic delirium.

Accordingly, the reports that Frenquel, without producing generalized nervous system depression, does in some specific way "block" or "erase" not only the psychic effects of LSD-25 but also delusions and hallucinations in acute schizophrenia have undoubtedly been received with considerable interest, or even surprise, by many psychiatrists.

II. Review of Previous Studies on the Psychological and Therapeutic Effects of Frenquel

The comments and criticisms made in the course of this review are based upon current literature on the methodology of drug evaluation and the author's own trials and errors in this field. A general review of methods of drug study and sources of error in their use has been given in a previous article (1).

Fabing has reported (2, 3) that premedication with Frenquel caused definite blocking of the psychological effects of 100 micrograms of LSD-25 or 0.4 gm. of mescaline sulfate. One hundred mg. of Frenquel given intravenously during the intoxication are said to have produced dramatic relief of LSD-25 effects within 15 minutes and apparently complete recovery within a half hour. The number of volunteer subjects used was small, and while several were unaware of the amount of Frenquel premedication given, the observers were not "blind". They may have been sub-

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[3] Frenquel: Merrell tradename for α-(4-piperidyl) benzhydrol hydrochloride. The Frenquel used in these studies was supplied by the Wm. S. Merrell Co.

ject to the bias of anticipating "blocking" effects. Moreover, subjects can vary widely in the extent of the psychic phenomena occurring or communicated during one LSD-25 trial as opposed to another, and one subject can differ from another in this regard. The small number of subjects and drug trials per subject fails to allow for this possibility. The importance of the personality structure of the subject, of his psychic state at the time the drug is given, and of the experimental setting as parameters conditioning observed drug effects has been previously emphasized (4).

Fabing has also reported (3) impressive results with Frenquel in patients with "schizophrenic reaction patterns," including schizophrenia *per se*, alcoholic psychosis, senile dissociation syndromes and post-operative confusional states. However, double-blind methods, placebo controls, systematic observational methods, or a design which controlled possible intervening variables were apparently not used in these studies.

These same limitations apply in general to the studies on Frenquel carried out by Proctor and Odland (5). However, these authors did report that 16 of 19 cases of delirium tremens showed complete relief of symptoms within 3–4 hours after receiving 20–200 mg. of Frenquel intravenously. In some cases the responses occurred within 30 minutes. These results are suggestive of a therapeutic effect from Frenquel, but unfortunately control cases and double-blind methods were not used.

Rinaldi *et al.* (6) have described a double-blind study of 16 weeks' duration on 39 chronically psychotic patients. The subjects were divided into two groups. One was on Frenquel for the second four-week period; the other, during the third period. The remaining periods were blocked out with Frenquel placebos. An extensive, descriptive psychiatric profile based on clinical observations was completed on each case by the investigators and attendants at the beginning, at the end of the second period, and at the conclusion of the study. Improvement ascribed to Frenquel was noted in 21 cases.

Excellent efforts at control were made in this study. However, rating scales or other efforts at quantification were apparently not used, nor does the report give the particular standards by which improvement was judged. Errors can arise in studies of this design after breaking the double-blind code, at the point of matching the summarized clinical evaluations with drug and placebo periods. The lack of precision in verbal descriptions of behavior is such that subtle degrees of selection, over-emphasis, under-emphasis, or relative weighing of the many aspects of recorded behavior can introduce significant bias.

III. Studies on Frenquel

The clinical trials with Frenquel to be described in this paper are not intended to be a definitive evaluation of the drug. They were actually carried out as exploratory pilot studies. They involved a relatively small number of cases and are by no means free of methodological defect. They would not be reported at this time except for the fact that the findings were remarkably consistent and in striking contrast to previous claims about the effectiveness of Frenquel. While the negative results in schizophrenics were not surprising, the questionable effects of the drug on toxic delirium prompted the author to repeat the experiments on the alleged capacity of Frenquel to block psychic disturbances induced by LSD-25. Here also the findings were very different from those described by Fabing.

A. Studies in schizophrenic patients

1. A double-blind study was carried out on five chronic schizophrenic patients, age 34–41, whose mental state was persistently characterized by delusional preoccupation and auditory hallucinations. Each patient was interviewed independently by two investigators five times each week for four weeks, and observations on his mental state recorded. During this period, starting at different times in each case, another physician introduced a two-week period of Frenquel administration. Each patient received identical placebos when not on the drug. The dose used was 40 mg. q.i.d.

While minor changes in mental state were recorded during the period of study, there was agreement by both observers that these bore no relationship to Frenquel administration. There was no change in the delusions or hallucinations of the patients. While the drugs

were given for a relatively short period of time, the dosage was higher than that reported to have had beneficial effects on such patients.

2. Individual case studies were carried out on 10 chronic schizophrenic patients, age 31–65, who were conspicuous for their delusions, auditory hallucinations, and in one case visual hallucinations. Each patient was seen daily in independent interviews by two psychiatrists. They were given Frenquel in doses of 20 to 80 mg. q.i.d. for periods varying from 10 to 40 days.

In none of the patients so treated, even with the 80-mg. dose, were significant changes noted in delusions, hallucinations, or mental status in general. Observations contributed by nurses and aides failed to reveal any significant changes in ward behavior.

3. Three schizophrenic patients, clinically similar to those treated in the above studies, were given repeated courses of Thorazine, Frenquel, and both drugs combined over a course of four months. Thorazine in doses of 50 to 200 mg. q.i.d. produced characteristic tranquilizing effects, and the hallucinations and delusional preoccupation of the patients, while still present, were less readily elicited in interview and less conspicuous in their ward behavior. The addition of Frenquel in a dose of 40 mg. q.i.d. to the Thorazine medication for periods of 10 to 20 days and its subsequent withdrawal produced no observable effects on their mental states. Frenquel alone when given to the same patients produced no changes in delusions, hallucinations, or general behavior.

4. A schizophrenic patient with incessant auditory hallucinations, which were readily verbalized as well as expressed in his behavior, was studied in an effort to determine what might be achieved with very high doses of Frenquel. He was given 50 mg. q.i.d. for seven days; the dose was then raised to 100 mg. q.i.d. for two days and finally to 200 mg. q.i.d. for three days! He was interviewed several times daily and carefully observed on the ward. He remained as agitated and hallucinated as ever. No sedative or tranquilizing effects could be detected even on the massive dosage of 800 mg. per day.

5. Three schizophrenic patients with auditory hallucinations and one with visual hallucinations were given 200 mg. Frenquel intravenously on repeated occasions without observable effects upon their hallucinations.

B. Studies on toxic delirium

1. Six patients with delirium tremens with characteristic visual hallucinations were treated. They were given 100 to 200 mg. of Frenquel intravenously. The patients were followed carefully, and the point at which hallucinations cleared was noted. In two cases the hallucinations could no longer be elicited four hours after treatment, but in four the hallucinations persisted for another 24 to 36 hours. One must suspect that the first two cases were nearing spontaneous recovery at the time treatment was given.

2. One patient with visual hallucinations associated with Korsakoff's syndrome and another with auditory hallucinations secondary to pathological alcoholic intoxication were not affected by 200 mg. Frenquel intravenously.

C. Studies on the blocking effects of Frenquel in LSD-25 intoxication

Five cooperative, well-integrated, convalescent schizophrenic patients were studied. The subjects were given repeated doses of 100 micrograms LSD-25 following premedication with Fenquel or Frenquel placebos. They were not informed of the drugs being given. The premedication dose of Frenquel varied from 10 to 40 mg. q.i.d. and was given for periods of two to five days. Matching trials with Frenquel placebos were carried out. The content and duration of the LSD-25 intoxications were followed by two observers. No evidence of blocking by Frenquel could be demonstrated.

In another series of experiments, subjects who had developed a characteristic LSD-25 intoxication from 100 micrograms of the drug were alternately given 100 mg. of Frenquel or a comparable volume of saline intravenously. Six trials were carried out in each of the five subjects. There was no evidence that Frenquel in the dose given changed the nature of the LSD-25 intoxication or shortened its duration when the results were compared with the saline controls.

A self-experiment on the effects of Frenquel on mescaline intoxication was carried out by

the author. The subject had ingested varying amounts of mescaline on previous occasions and was familiar with the effects of that drug. Forty mg. of Fenquel q.i.d. were taken for three days before ingesting 0.2 and 0.4 gm. of mescaline sulfate. The intoxications which ensued were identical in all respects to that which had been previously experienced from these doses of mescaline without Frenquel premedication.

Summary

Treatment of schizophrenic patients with oral and intravenous Frenquel failed to confirm claims that this drug has "anti-delusional" or "anti-hallucinatory" effects on such cases. The drug was of no therapeutic value in the patients treated. Even large doses did not modify their behavior or mental symptoms in any consistent way.

A small series of patients with toxic delirium was treated with intravenous Frenquel. No evidence was obtained that the drug has predictable or consistent effects upon the hallucinations or other mental abnormalities associated with such mental disturbances.

Studies on the effects of Frenquel on LSD-25 intoxication in experimental subjects failed to confirm reports that premedication with this drug "blocks" LSD-25 intoxication or that it exerts a specific suppressive effect when given intravenously at the height of the intoxication. In self-experiments with mescaline sulfate, the author found no subjective evidence that premedication with Frenquel modifies the nature or duration of mescaline intoxication.

BIBLIOGRAPHY

1. Clark, L. D.: Evaluation of the therapeutic effects of drugs in psychiatric patients: Sources of error and appraisal of investigational methods. *Dis. Nerv. System* (To be published).
2. Fabing, H. D.: New blocking agent against the development of LSD-25 psychoses. *Science, 121:* 208, 1955.
3. Fabing, H. D., and Hawkins, R. J.: A year's experience with Frenquel in clinical and experimental schizophrenic psychoses. *Dis. Nerv. System, 16:* 329, 1955.
4. Lindemann, E., and Clark, L. D.: Modifications in ego structure and personality reactions under the influence of the effects of drugs. *Am. J. Psychiat., 108:* 561, 1952.
5. Proctor, R. C., and Odland, T.: Frenquel. *Dis. Nerv. System, 17:* 25, 1956.
6. Rinaldi, F., Rudy, L. H., and Himwich, H. E.: The use of Frenquel in the treatment of disturbed patients with psychoses of long duration. *Am. J. Psychiat., 112:* 343, 1955.

THE EFFECTS OF CORTISONE ON LSD-25 INTOXICATION IN SCHIZOPHRENIC PATIENTS[1]

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Introduction

It is well known that in some patients cortisone administration can cause prolonged episodes of psychotic behavior. An extensive review of the literature on cortisone mental disturbances has been carried out by Quarton *et al.* (2). A satisfactory explanation of these disturbances has not yet been achieved. The mental effects of cortisone are about as unpredictable in schizophrenics as they are in mentally normal patients (1).

Lysergic acid diethylamide (LSD-25)[4] has been used extensively as a research tool because it is capable of producing abnormal states of mind which simulate many of the phenomena of the major psychoses. It has been reported by Savage (3) that schizophrenic patients are less reactive to LSD than normal subjects. It would be important to know whether such difference in reactivity is a matter of pharmacological sensitivity to LSD or an apparent difference based upon the extent to which the psychological effects of the drug are communicated to the observer.

Cortisone is a potentially psychotogenic substance while LSD in adequate dosage is consistently psychotomimetic. Accordingly, it was felt to be of theoretical interest to determine what effects, if any, intensive premedication with cortisone might have upon the sensitivity of schizophrenic patients to varying doses of LSD. In order to do this, it was necessary to establish a baseline in terms of the response of each subject to LSD. At the same time, an impression would be acquired of the sensitivity of these schizophrenic patients to LSD.

Experimental Design

A. Subjects: Five young schizophrenic patients with normal physical conditions were employed in this study. All were passively cooperative, well adjusted to hospital routine, moderately well-compensated in terms of their psychoses, and had been relatively stabilized in mental state for several months prior to the study. Although all would respond appropriately to routine questions, they did vary from one to another in their capacity or willingness to talk about personal concerns or to describe in detail their mental or emotional state. Subject 5 was particularly reticent in this regard.

B. Method: During a 40-day control period, the subjects were given oral doses of LSD at intervals of 3–4 days. The starting dose was 10 μg. The dosage was increased by 5–10 μg. at each successive trial. The subjects were interviewed by two psychiatrists at a constant time after the ingestion of LSD. Further observations on their behavior were contributed by nurses and aides on the research ward. The subjects were not aware of the nature of the drug being given.

The dosage was increased until the visual disturbances characteristic of LSD intoxication were induced. These perceptual phenomena were chosen because the point at which they appeared was more definite than the other effects of LSD. The visual disturbances consisted of colorful sparks of light, varied coruscations, elementary visual patterns in the form of geometric designs, and a variety of illusory phenomena by which objects in the environment appeared wavy, distorted, etc. The presence of these phenomena was also frequently reflected in the general behavior of the subjects, who would stare persistently at walls or ceilings unless distracted. The dosage necessary to produce these visual effects in a given subject was

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[4] The LSD-25 used in this study was generously supplied by Sandoz Pharmaceuticals.

arbitrarily designated as his LSD threshold. Raising the dose 10–20 μg. above the threshold level consistently produced the visual disturbances in more marked form.

The subjects were then started on cortisone in a dose of 50 mg. q.i.d. After 6 days of cortisone administration, the threshold dose was repeated and the effects observed. Further trials were then carried out in each subject with doses varying from 10–30 μg. above and below the threshold dose level.

Observations

A. A reasonably definite threshold dose of LSD could be determined in 4 of the 5 subjects. This was 70 μg. in 2 subjects, and 60 and 80 μg. in the others. The fifth subject, however, over the course of 15 trials in doses from 10–130 μg. never admitted any effects from LSD other than feeling "drowsy." No significant changes in his behavior were noted until he was given 120 μg. On this occasion, he stared intently at the ceiling and repeatedly blinked or brushed his hands over his eyes as if to clear his vision. Hallucinations were suspected by the observers but not admitted by the subject.

Sub-threshold doses of LSD in subjects 1–4 produced a variety of minor effects. They were generally more alert and mildly euphoric but physically underactive. There was some smiling and giggling. They described themselves variously as feeling "drunk", "hung-over", "numb", "dopey", "dizzy", "confused", or on "Cloud 8". Some psychological effect was evident even with the smallest doses. None of the subjects became uncooperative, hostile, or showed any serious behavioral disturbances. They appeared to have no difficulty distinguishing the LSD intoxication from their other mental difficulties. The major effects of LSD subsided in each instance within 4–6 hours.

B. No significant changes in sensitivity to LSD were observed in any of the subjects when they were studied while on maintenance doses of cortisone. The threshold doses of LSD as well as doses above and below this level produced the same effects as before.

C. Fifteen days of cortisone treatment did not produce significant changes in the mental state of the subjects studied.

D. Despite the impaired capacity for communication inherent in their schizophrenic illness, the account of the subjective effects of LSD given by these patients did not differ appreciably from that of many normal subjects of limited basic verbal skill and intellectual attainments. The descriptions of LSD intoxication offered by such subjects are admittedly meager when compared with those which have been given by some of the bright, introspectively curious, and verbally resourceful "normal" volunteers whose accounts occupy a conspicuous place in the literature on hallucinogenic drugs. It was noted in this study that the elaborateness of the description given of the LSD intoxication was related directly to the usual communicative ability of the subject. Because one is so dependent upon introspective reports for evaluating LSD effects, reluctance or inability to communicate subjective experience may be construed as lack of effects. The impression from this study is that schizophrenic patients do not differ from normals in their pharmacological sensitivity to LSD.

Summary

In the schizophrenic patients studied, premedication with cortisone had no observable effect upon their sensitivity to LSD intoxication. It is felt that schizophrenics do not differ from normal subjects in their pharmacological sensitivity to LSD, but may differ in their capacity to communicate the subjective aspects of the LSD experience.

BIBLIOGRAPHY

1. Lewis, A., and Fleminger, J. J.: The psychiatric risk from corticotrophin and cortisone. *Lancet 1:* 383, 1954.
2. Quarton, G. C., Clark, L. D., Cobb, S., and Bauer, W.: Mental disturbances associated with ACTH and cortisone: A review of explanatory hypothesis. *Medicine, 34:* 13, 1955.
3. Savage, C.: Variations in ego feeling induced by D-lysergic acid diethylamide (LSD-25). *Psychoanal. Rev., 42:* 1, 1955.

CARBON DIOXIDE THERAPY

A Clinical Evaluation of 200 Patients

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During the past few years carbon dioxide has been used in the treatment of mental disorders both here and in Europe. Interest was aroused by Meduna who first gave it to neurotics in 1946 and subsequently published several papers and a book which appeared in 1950 (6).

The treatment is simple to administer if proper precautions are observed. The patient inhales a mixture of 30 per cent CO_2 and 70 per cent O_2 through an anesthesia mask until a state of brief coma (lasting less than one minute) is reached. The mask is then removed, and the patient rapidly regains consciousness. He may feel slightly dizzy for a few moments. No preparation or restraint is necessary, and rapid recovery always occurs. Since the patient receives excess O_2 throughout, there is no risk of cerebral anoxia.

Meduna recommended three treatments per week to a minimum of 20 treatments. He purposely omitted psychotherapy. He found that about 62 per cent of 100 neurotics improved, after excluding a group of obsessional neurotics who did not respond. He claimed that CO_2 is a "specific" therapeutic agent for psychoneuroses.

METHOD

The present study was begun in 1949 to reinvestigate Meduna's claims. Half of the patients were treated at Vanderbilt Clinic (Series C) and the other half in a private office (Series P). Series C consisted of 100 unselected cases, most of whom were severely disturbed neurotics, plus a few ambulatory psychotics. Most of them had not heard of carbon dioxide therapy (CDT), and many had failed to respond to previous psychiatric or medical treatment. They were usually referred by outside physicians or from other clinics at the hospital. None of this group received N_2O induction or concomitant planned psychotherapy.

Series P also consisted of 100 unselected patients, many of whom had previously read or heard about CDT and requested it. This group tended to be more cooperative and better educated than Series C. Concomitant psychotherapy was used in 15 patients and N_2O induction in 29. The therapeutic environment was quite different in the two groups: Series P patients had only one therapist throughout, whereas those in Series C were treated by two or three therapists.

The multiple-breath technique of Meduna was used, most patients receiving treatment three times weekly. It was not possible to set up adequate controls, hence each patient served as his own control.

Information was obtained from patients by personal interviews, questionnaires, and the Jackman-Schorr Neurotic Index (4). The data were transferred to Peck's standardized punch-card (9), and a simple statistical evaluation made. We followed the APA classification of mental disorders (7). In cases where more than one diagnosis was necessary, only the major diagnosis was used in classification. Percentages were calculated only for descriptive purposes, the sample being too small to give precise values.

In a critical review of the literature (2) we discussed fully the difficulties encountered in evaluating CDT, and hence will not repeat them here. We attempted to be as objective as possible in reporting results, despite the fact that no independent observers checked the results, and follow-up studies were incomplete. The term "improved" indicated relief of major symptoms occurring *during* CDT which were noted both by patient and therapists. Changes in behavior were not rated separately, but many patients observed improvement in work, social or sex life which they attributed to the treatment. Patients who became worse or showed minimal or no improvement were classified "unimproved".

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TABLE 1.—GENERAL DATA

Series	Total	Sex		Age					Severity			No.	CDT
		M	F	11–20	21–30	31–40	41–50	50+	Mild	Mod.	Sev.	1–10	11+
C	100	57	43	2	30	38	18	12	3	37	60	48	52
P	100	56	44	1	25	28	27	19	3	36	61	34	66

TABLE 2.—NUMBER OF CDT VS. IMPROVEMENT

No. CDT	200 Cases	Improved	
		No.	Per Cent
1–2	35	0	0
3–10	47	9	19
11–20	30	12	40
21–35	30	14	46
36–60	45	32	71
over 61	13	11	84

TABLE 3.—IMPROVEMENT RATES

	200 Cases		118 Cases	
	C	P	C	P
Greatly improved	2	7	2	6
Improved	27	42	26	35
Unimproved	65	48	22	23
Worse	6	3	3	2
Per cent improved	29%	49%	53%	62%

RESULTS

Table 1 gives general data about each group. It will be noted that the sex distribution was equal in the two series. A slightly larger number of cases fell in the 31 to 40 year age group in Series C than in Series P. There was a similarity in severity of illness in both groups. More patients in Series C stopped before receiving 10 treatments than in Series P. Patients who received less than 10 treatments showed no significant change. Many patients (41 per cent of the entire group) stopped before receiving 10 treatments, usually against the advice of the therapists. This can be reduced by N_2O induction, which we now use routinely. It is harmless if certain simple precautions are observed.

For statistical accuracy, any patient who received one or more CDT was included in the over-all improvement rate. From Meduna's standpoint (6), patients who received less than 20 CDT would be considered "untreated" since he advocated a minimum of 20 treatments before determining whether or not improvement had occurred. At the start of this study, however, we had no preconception as to the minimum number of treatments required. A group of 118 patients who received more than 10 treatments was selected for detailed statistical evaluation. This will be referred to as the "treated" group.

Table 2 shows improvement in relation to number of treatments. A positive correlation was obtained in that patients who received more than 35 treatments showed as high as 84 per cent improvement. This might be accounted for as an "automatic" selection since patients who felt better during the early part of the treatment would tend to stay with it the longest. On the other hand, recent findings by Schaeffer (11) indicate that there appears to be a cumulative effect of carbon dioxide on the nervous system, which he observed in animal experiments and in humans. He believes that this may account for the large number of CDT necessary to achieve improvement.

Table 3 compares improvement rates of the total group with the 118 treated cases. It will again be seen that a much higher degree of improvement was noted in those patients who had more than 10 treatments. Our over-all improvement rates are lower than most workers in this field. However, the rate of 62 per cent in "treated" cases of Series P is identical with that noted by Meduna in his original 100 cases. It is of interest that in the review of 238 cases selected from the literature to 1953, the over-all improvement rate was 61 per cent. The individual range was very great, however, from 22 per cent to 93 per cent (2).

A relationship was found between duration of illness and improvement. Patients sick less than 3 years had a 62 per cent rate, while those sick more than 10 years had 51 per cent.

Table 4 shows diagnosis related to improvement. Anxiety and conversion reactions had a 70 per cent improvement rate, whereas hypochondriacal, obsessive-compulsive, and psy-

chotic reactions showed less than 50 per cent. The 60 per cent rate in depressive reactions was considerably higher than most other workers have found with CDT.

No correlation was found between improvement and previous psychiatric treatment, age, or sex. The few patients in Series P who received CO_2 plus N_2O had a higher rate than those who received CO_2 alone.

In a separate study of 86 patients with anxiety reactions of various types we found a 68 per cent over-all improvement rate (3). The maximum response occurred in those who had symptoms for less than three years. CDT offers a method of "sorting out" three types of patients with anxiety reactions. The *first* group exhibited extreme tension and anxiety after the first treatment and immediately discontinued the treatment. The majority described panic or a feeling of suffocation. The *second* group showed treatment anxiety of a less marked degree but requested continuation of treatment. The *third* group noted decrease of anxiety after a few treatments and thereafter maintained steady improvement. This group showed maximal recovery. It was not possible to predict into which group a patient would fall.

CDT would appear to be the most innocuous form of physical therapy in psychiatry today, though it is not entirely free from risk. A man in Series C, with right bundle-branch block, who received one non-coma CDT, stopped and several months later wrote that "the treatment brought on a heart attack". This was not confirmed medically, and he was in good health at the time of writing. Another patient fell off the table during an excited abreaction but did not injure herself. A female schizophrenic in Series P made a suicidal attempt after 10 CDT. This patient had been in psychoanalysis for several months and had made previous suicidal attempts. She made an uneventful recovery. We agree with certain investigators (1, 8) that CDT should be used with extreme caution in "borderline" psychotics because of the risk of precipitating a psychosis. Supportive psychotherapy and EST are preferable in severe depressive reactions. Several patients complained of transient pains in the chest and abdomen during and immediately after the treatment.

There were many patients (41 per cent) who stopped against advice. The following comments were typical of this group: "I couldn't breathe and didn't like the mask over my face." "My relatives were gassed by the Nazis, and I had frightening dreams during the treatment that I would be gassed also." One schizophrenic reported: "CDT makes me feel even more in a dream-world than usual." "It touches what is bothering me, but it frightens me."

TABLE 4.—DIAGNOSIS VS. IMPROVEMENT

Diagnosis	118 Cases	Improved	
		No.	Per Cent
Anxiety reaction	44	31	70
Conversion reaction	10	7	70
Impotence	3	2	66
Depressive reaction	10	6	60
Phobic reaction	12	7	58
Hypochondriacal reaction	8	4	50
Miscellaneous reaction	13	6	46
Schizophrenic reaction	9	4	44
Obsessive-compulsive reaction	7	3	42
Manic-depressive reaction	2	0	0

On the other hand, dramatic and unexplained improvement was shown by some patients after a few treatments. One man with severe constant asthmatic attacks of 4 years' duration received three CDT and wrote several weeks later that he had had no further asthma attacks and felt that he was cured. The follow-up was not long enough, and this may have been a spontaneous remission. It is of interest that patients in Series P who received more individual attention and were subject to less environmental change tended to respond in general in the same way as the clinic group although a smaller number of the former dropped out.

Psychological factors play a major role in the therapy. Transference reactions and abreactions of a bewildering variety were observed. A few patients developed "psychic addiction" to the treatment and had to be gradually weaned from it. A therapist in Series C was quite aggressive in his approach, and on days when he gave the treatment, only one or two patients appeared. Two days later, when a well-liked therapist was present, 12 patients showed up. A form of group therapy was noted, patients "comparing notes" outside the treatment room and reassuring one another.

Several female patients described orgastic sensations during the treatment, and some made erotic advances to the therapists while coming out of it. Dreams of all types occurred during the period of coma, and one patient had a repetitive identical dream during each of 35 treatments in which he was in a baseball game and made a home-run. Patients who requested the treatment or who were convinced it would help them showed the highest degree of improvement.

It was of interest that a small group of patients who had had various types of psychiatric treatment previously without responding, and who were very resistant to CDT, nevertheless showed marked improvement.

There are few contraindications to CDT: advanced heart or lung disease, severe hypertension or acute infections. Nitrous oxide must be used with caution, but even in patients sensitive to it, the stimulating effect of the subsequent CO_2O_2 mixture upon the respiratory center will counteract any cerebral anoxia. Recently a patient had a fatal coronary thrombosis during CDT (10). No other fatalities have been reported, though many thousands have received this therapy.

Comment

Meduna stated that the effects of CDT are "purely physical" and developed an elaborate neurophysiological theory that CO_2 interrupts pathological hypothalamic-frontal reverberating circuits by raising the threshold of stimulation of the higher nerve centers. About ten other theories have been proposed (2). Some psychoanalysts believe beneficial effects are due entirely to "suggestion" and that masochistic or other unconscious needs may be satisfied by CDT. We do not believe that our results can be explained purely as suggestion since the therapists played a minor role, giving a minimal amount of reassurance and *not* stressing that CDT would help. CDT may benefit some patients. However, we feel that ideally it should be combined with and followed by psychotherapy to maintain lasting improvement through the development of insight. This is impossible from a practical point of view and can be done only in selected cases.

It is well known that neurotic individuals tend to respond to almost anything that is done to them. Landis (5) made a study of neurotics discharged as improved from state mental hospitals from 1925–1933. He found 68 per cent improvement and claimed that any method of treating neurotics must show a higher rate to be considered "specific." However, all of his patients received at least custodial care, which is a form of "treatment." It is of interest that almost every form of treatment, from psychoanalysis to CDT, claims 60–70 per cent efficacy in the neuroses. One might question, therefore, whether our rate of 62 per cent with CDT is significant. As with any new treatment, a storm of controversy has arisen, and it will take many years to validate CDT accurately.

Conclusions

Our studies tend to confirm Meduna's that CDT is of value, when used without planned psychotherapy, in the treatment of psychoneuroses, particularly anxiety and conversion reactions. It would appear to be of little value in hypochondriacal, obsessive-compulsive, depressive reactions and "borderline" psychoses. We do not believe that it is "specific" or necessarily superior to psychoanalysis, psychotherapy, or various somatic treatments currently being used in psychiatry. Since it is physically harmless, easy to give, inexpensive, and occasionally highly effective, it deserves to be studied further.

CDT can be readily misused by unqualified or unscrupulous people who charge high fees and promise miraculous results. For this reason definite safeguards are essential. It should be administered only by qualified psychiatrists or other physicians specially trained in the treatment of emotionally disturbed patients.

BIBLIOGRAPHY

1. Carbon dioxide treatment of psychoneuroses and modifications thereof. A Symposium held at Atlantic City, New Jersey, Committee on Carbon Dioxide Research, May, 1952.
2. Frank, J. A.: A critical evaluation of carbon dioxide oxygen inhalation therapy in mental disorders. *Am. J. Psychiat., 110:* 93–103, 1953.
3. Frank, J. A.: Carbon dioxide therapy (CDT) in anxiety reactions. (To be published.)
4. Jackman, A. J., and Schorr, C. A.: Evaluation of

carbon dioxide treatment of neuroses. *J. Clin. & Exper. Psychopathol., 13:* 31, 1952.
5. Landis, C.: *Concepts and Problems of Psychotherapy,* Chapter V, Hinsie, L. E. New York: Columbia University Press, 1937.
6. Meduna, L. J.: *Carbon Dioxide Therapy.* Springfield, Ill.: Charles C. Thomas, 1950.
7. *Mental Disorders: Diagnostic and Statistical Manual.* Washington, D. C.: American Psychiatric Association, 1952.
8. Milligan, W. L.: Treatment of psychoneurosis; Modified CO_2 abreactive technique. *Brit. M. J., 1:* 1426, 1951.
9. Peck, R. E.: Standardization of carbon dioxide treatment. *Dis. Nerv. System, 14:* 240, 1953.
10. Reported at Meeting of Committee on Carbon Dioxide Research, Atlantic City, New Jersey, May, 1955.
11. Schaeffer, K. E.: Adaptation of men and animals during prolonged exposure to increased carbon dioxide concentration. *Am. J. Physiol., 163:* 747, 1950.

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MOTHERS OF SCHIZOPHRENICS[1]

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In this preliminary note, I should like to state and superficially examine the proposition that the mothers of persons who become schizophrenic have special psychological characteristics.

No attempt will be made in this note to review the literature on this subject, not even the major contributions of Federn, Sullivan, Fromm-Reichmann, Kasanin, Winnicott, Bender, Rosen, and Sechehaye.[2]

Many schizophrenic adults, in one way or another, fail to come to livable terms with their mothers and continue to suffer, to a very marked degree, with feelings coming from the relationship between the sufferer and the mother. The nature of the feelings can be simplified as their extremes: some schizophrenic people continue to be the helpless and willess infants of their mothers; some murder their mothers. Both of these extremes are more often symbolized in psychotic ideation and symptoms than acted out in life. More commonly than the extremes is seen the ambivalent mixture of both; the individual who relates to his mother in a sort of frightful symbiosis, struggling with rapidly alternating and very powerful wishes for total love and total destruction of the mother.

Several workers with great experience in the treatment of schizophrenia have directed their attention to the possibility that much of the genesis of the schizophrenic psychosis can be traced back to the nature of the relationship between mother and child in the very early part of the child's life.

It seems to me that the mothers of schizophrenics, when I have been able to know them a little, have in common certain delusions having to do with sexual intercourse, pregnancy, childbirth and babies. These delusions are not limited to the mothers of schizophrenics; but they do, in these women, combine to form a specific result. This result is a very specific power-operation, a method of secretly controlling the child.

The women whom I have studied have poor opinions of their own worth. Many of them are seen during the time when their child has been hospitalized with an acute psychotic episode, and at such times, they are much concerned with the idea that they have failed as mothers; that if they had been good mothers, their children would not have psychiatric disease. This, of course, is not specific for the mothers of schizophrenics, but is generally found in many parents of children with any kind of psychiatric (and other) disease.

The mothers of schizophrenics readily express doubts of their own worth going far back in life. These mothers seem to get little or no pleasure from being women. The usual feminine functions, biological and social, are carried out with little interest, little satisfaction, or else are avoided. Sometimes compensatory activities are invested with enormous amounts of energy. Thus, the mother of one schizophrenic patient averaged a full working week, forty hours, playing bridge. Another mother was very busy all week practicing law, reserving Sundays for her child.

Sometimes the social facade presented by the patient's mother makes it difficult for a long time for the psychiatrist to get at her basic feelings about her child. One such mother impressed me over a period of about six months as a warm, sympathetic, secure and giving woman. I first began to understand her need

[1] Read at the meeting of the Western Regional Conference of the American Psychiatric Association in San Francisco in October, 1955.

[2] After this paper had been written, Dr. Lewis Bernstein of the Veterans Administration Hospital in Denver, Colorado, called my attention to a paper written by Dr. Joseph C. Mark of the Bronx Veterans Administration Hospital entitled "The Attitude of the Mothers of Male Schizophrenics Toward Child Behavior." There is in this paper a review of several articles in the psychological literature, namely: Gerard, D. L., and Siegel, J.: The family background on schizophrenia. *Psychiat. Quart., 24:* 47–73, 1950; and Tietze, T.: A study of mothers of schizophrenic patients. *Psychiatry, 12:* 55–56, 1949. The review contains material pertinent to this issue. Furthermore, Dr. Mark's paper contains conclusions, based on experimental methods, about feelings which the mother of a schizophrenic has for her child. These conclusions are somewhat similar to certain points in my hypothesis.

to control her child when, on one occasion, the daughter did not put on a jacket in a drafty theater at the mother's suggestion. The mother reached into the girl's sleeve and with her fingernails inflicted four lacerations on the girl's breast.

Of the eight mothers included in this study, seven considered themselves to be inadequate sexual partners, although the feeling of inadequacy was usually weakly defended by projection or rationalization. They all had the idea that "successful sexual adjustment" is the one and absolute criterion for the goodness or badness of the relationship between a man and a woman. Ideas about companionship, mutual service, and shared goals are lacking.

In two of these women, there could be demonstrated, as a basic difficulty, strong penis envy with the delusion that the penis envy could be permanently satisfied by the possession of a baby.

Three others had no apparent masculine striving but felt incomplete as women; they felt that the permanent possession of a baby would make them complete.

After the birth of their babies, six of these mothers wished that the babies had been of the other sex (four girls, two boys). One occasionally dressed her son as a girl and called him by a girl's name as late as his ninth year.

We admitted to the hospital a 36-year-old schizophrenic man who had been accustomed to staying home and wearing women's clothing. When it became apparent that he would have to be hospitalized, his mother borrowed a pair of trousers for her son to wear.

All of these mothers seem to me to have had unusually difficult pregnancies. Apparently all of them had been rather ignorant of the physiology of pregnancy although this is obscured by the fact that the most recent account was 18 years after the child's birth. All had great fear of delivery, and this fear seems to have been of the general nature of some serious and irreparable structural damage. One woman feared her vagina would be enlarged and her ability to enjoy intercourse thereby permanently impaired. Three were fearful and preoccupied with losing their figures, specifically, permanent enlargement of the abdomen and sagging of the breasts. The ideas of structural damage in childbirth were merged with the idea of being inadequate as a sexual partner: frigidity and/or the husband's disinterest being blamed on the bodily changes following childbirth.

The damage of child-bearing tended to be blamed in a very personalized way on the baby, as if the baby had willed to damage the mother and steal away her sexuality. This suggests a post-partum version of the sadistic parasite fantasy of pregnancy.

As a continuation of these ideas, five of the eight mothers feared death in childbirth. This fear seemed to me to be much in excess of normal. A corollary to this was a rivalrous fear of being excluded, by death, from continuing relationship to the husband, or less often, to the child. To be more specific, they feared that the husband would be left in the possession of the child. All eight of these women complained that their husbands were inadequately thoughtful or attentive or sympathetic during the pregnancy. I cannot quantitatively compare this to normal, but I suspect that it is in excess of normal.

In their memories of the schizophrenic patient's childhood, not one of these mothers described the usual mixture of desirable and undesirable characteristics. Six described little angels, who were never naughty, who were toilet-trained early and easily, who were intelligent and tractable, even gifted. One child read the newspaper and did mental arithmetic at the age of four, another played the piano at four, another was completely toilet-trained within one week after the training began.

The other two mothers described wicked children, who cried unreasonably, were demanding and intractable.

The strongest impression I had from these mothers was that they continued to control their children for a very long time. The impression was always arrived at indirectly; the mothers described vividly and unmistakably the control which they exercised in fact, but they themselves apparently never perceived or admitted consciously the control. The same control was described by the patients themselves. The control was variably consciously realized by the patients. In periods of psychotic excitement, there was usually

understanding of the fact of maternal control, associated with enormous conscious hostility.

One aspect, then, of the control was its secrecy. It would seem that the control was not consciously perceived either by mother or child until the time of frank psychosis, when it was perceived by the child.

The control has other characteristics. It is almost incredibly extreme in degree. The mother attempts to control not only the behavior of her child but also his thoughts, wishes and bodily functions. Thus, one mother subjected her daughter to interrogations after her dates sometimes lasting two hours. This mother had previously questioned her daughter at great length about the happenings at school. The same method was used for the therapy which took place between the daughter and me after she became ill. This mother wished also to question me in great detail about the interviews between her daughter and me.

Interference with vegetative functions was a routine procedure among five of these mothers: enemata, sedatives and inducing vomiting.

The other characteristic of the control is that its main powers are derived from appeals to pity, guilt and shame in the child. The premise is very early established between mother and child that the mother has suffered greatly and sacrificed greatly for the child; the child is very deeply indebted to the mother. The child has the duty of repaying the debt with gratitude and obedience. Anger or rebelliousness is specifically forbidden. It goes without saying that not all children react in the same way in such a situation; not all react passively, and almost none reacts consistently.

I offer, then, the hypothesis to be investigated that in the relationship between a mother and her schizophrenic child, the following characteristics are common: 1) The mother does not enjoy being a woman. 2) The mother wishes continuing control of her child. 3) The control is a) secret, b) extreme in degree, c) acquired by manipulation of pity, shame and guilt.

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PSYCHOSIS FOLLOWING LUMBAR PUNCTURE

A Case Report

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Removal of cerebrospinal fluid by means of lumbar puncture is generally an innocuous procedure, although it may be attended by some complications in occasional instances. The most common such complications are post-lumbar puncture headaches, introduction of infection, and herniation of the brain stem due to release of increased intracranial pressure. The purpose of this paper is to discuss the implications of a psychotic reaction following a lumbar puncture.

Although Dana (2), as early as 1917, believed that post-lumbar puncture headaches more frequently occurred in "nervous people", few reports have appeared in the literature describing psychopathological reactions to this diagnostic procedure. Thus, Dripps and Vandem (4) and Glesne (5), in recent rather comprehensive papers, do not mention psychological factors. Redlich, Moore and Kimbell (9) conclude that "drainage is the most significant factor in the production of symptoms following lumbar puncture, outweighing by far the small contribution of anxiety, hypochondriasis and other emotional elements." Their impression is based on a statistical analysis of a group of patients who were "tapped" with needles of different gauge. In addition to grouping their subjects according to the size of spinal needles used, the authors also classified their subjects on the basis of emotional stability. They found only a slight increase in symptoms in the unstable group. Since this group was fairly heterogeneous, it is possible that more significant results would have been obtained if the authors had considered the meaning of the procedure to the subject, the subject's neurotic reactions to pain, or other more specific psychodynamic factors.

Other than occasional reference to psychogenesis of lumbar puncture headaches (2, 3, 8), no references to any other disturbance arising on a psychogenic basis from this procedure could be discovered in the literature of the past 20 years. Such authoritative texts of neurology as Wilson (10), Brain (1), and Grinker (6) made no mention of untoward psychological reactions to lumbar puncture. This may be the result of the rare occurrence of such complications, or it may be attributed to the fact that the reactions were not noted, or that they were related to some other factor by the observers. In the case to be presented, a clear relationship between the performance of a lumbar puncture and the development of a psychotic episode was seen.

Although any medical procedure involving instrumentation or penetration of a body cavity may arouse anxiety, confidence in the physician serves considerably to mitigate this reaction. Other than fear of the potential physical danger and anxiety about the unfamiliarity of the procedure, there is another source of psychological danger inherent in some medical investigative procedures. This is dependent on the patient's reaction to the symbolic meaning of the procedures. Thus, any surgical operation may arouse pathological reactions as a result of mobilizing castration anxiety. Digital examination of the prostate gland or rectal temperature recording may stimulate considerable homosexual anxiety in susceptible individuals. Dental surgery may be traumatic to persons with oral conflicts (7) and anesthesia to others with hysterical problems.

In the light of the foregoing remarks, a case report of a major psychological reaction following a lumbar puncture is presented.

CASE REPORT

A 35-year-old petty officer in the United States Navy was transferred to a U. S. Naval Hospital for observation with an anxiety reaction. For an indefinite period prior to hospitalization the serviceman became progressively depressed, anxious, and preoccupied with marital problems and an impending divorce from his wife. A week prior to his

admission to the hospital, the patient became suspicious of his shipmates and thus aroused the concern of the ship's medical officer. His suspicions were in response to good-natured "kidding", to which the patient was exposed because he assisted a woman of questionable repute in a fight with her husband. The patient reacted as if he had been accused of some misdemeanor and felt that his shipmates would prejudice his chances of reunion with his own wife. No other suggestion of delusional thinking was noted at that time, and his reaction to the teasing was not of sufficient intensity to be classified as delusional.

Upon admission to the hospital, other pertinent symptoms, such as slight circumstantiality and suggestive deficiencies in abstract thinking and judgment, were noted by the admitting psychiatrist. The serviceman's record indicated that he had done well in his Naval career, had been able to assume responsible assignments and had no previous history of psychiatric disorder. For these reasons, and since there was considerable stress in the precipitation of the current illness, it was anticipated that supportive psychotherapy of brief duration, along with some attempts to clarify the marital situation, would suffice to enable the patient to return to duty. Under this regimen the patient gradually improved during the subsequent two weeks. A rather dependent relationship with some latent homosexual implications was quickly established by the patient. He was friendly, at times almost familiar, highly cooperative and obsequious. Gradually he began to feel that his symptoms were the result of his marital problems, and he became free of any suggestion of delusional thinking or excessive suspiciousness.

In order to complete the medical workup, and in view of the presence of questionable sensorial and intellectual deficiency on the mental status examination, further studies were done to evaluate the possibility of organic brain disease. Electroencephalographic and roentgenologic studies were performed without event and revealed normal findings. A lumbar puncture was performed by the patient's medical officer easily and without mechanical difficulty. Prior to the lumbar puncture, the patient had experienced some apprehension but was easily reassured. Immediately following the procedure, the patient smilingly said that it was not nearly as bad as he had anticipated and that he almost enjoyed it. There was no post-lumbar puncture headache, and the results of laboratory and manometric determination were within normal limits.

The medical officer was ill for the next week, and the patient did not appear for his regular appointment when he was informed of the return of the doctor. Within four days after the spinal tap, the patient had become progressively apprehensive, withdrawn and suspicious. He was deluded in thinking that his medical officer was to have him court-martialed because he had struck the medical officer. He went so far as to try to enlist another patient as a witness to the fact that he did not strike his doctor, but merely gave him a friendly pat on the arm following the spinal tap.

During the next few interviews, the patient remained quite tense, apprehensive and preoccupied with the conviction that he would be punished for hostile attacks on the doctor. There were some depression and suicidal preoccupation observed, as well as considerable withdrawal from ward activities. An outstanding feature was the patient's certainty that he had not been sufficiently approachable and friendly. He was markedly self-critical because of this feeling of aloofness. He would then refer to his behavior at the time of the lumbar puncture, and he said that the doctor *must* have thought his attitude was hostile. He requested of the therapist that he "take me into a quiet room (isolation room) and work me over." After this request, he stated he had "no murderous impulses, especially none toward you." During these interviews the patient would frequently pull or scratch at his genitals through his clothing. He would also move his chair about so that it would end up close to the doctor's chair. There generally was reluctance to leave the room at the end of an interview, and on several occasions the patient took objects of no intrinsic value with him. During this period it was found that he would improve to some extent if the therapist maintained a distant, cool attitude.

The past history was somewhat incomplete since reliable information could not be obtained from the patient and his family lived a considerable distance away. The patient's birth

and early development were uneventful according to the parents, and psychological symptoms were not noted until the recent episode. Notwithstanding his shyness as a youngster, the patient was popular and had many friends. He was close to his family whom he assisted on the farm after school hours. The patient completed high school with good grades in 1940 and then entered the U. S. Navy. About 1950 he married a woman with whom he lived for only a few weeks before going overseas. When he returned, he discovered that she had been having an affair with another man, and the patient obtained a divorce on the grounds of infidelity. During the summer of 1954, the the patient met his second wife whom he married six months later. They lived together for only five weeks, following which the patient again reported for sea duty. There was a good deal of marital disharmony characterized by sexual incompatibility. The wife complained of the patient's excessive sexual demands, and he complained of her lack of interest. The wife felt that the patient was abnormally jealous of her two and one-half year old daughter by a previous marriage. After he left for sea duty, the patient and his wife had an initially affectionate correspondence, but her letters gradually became indifferent and cold and culminated in her request for a divorce. The patient suspected that another man was involved in his wife's decision to leave him.

Discussion

The case presented is of a serviceman who was referred to the hospital because of anxiety and suspiciousness, but who was able to reintegrate with the assistance of superficial psychotherapy to the point of being well enough to return to duty. Following a lumbar puncture, performed by his psychiatrist to rule out the possibility of organic brain disease, the patient was apparently precipitated into a severe paranoid schizophrenic episode.

One must briefly consider the possibility of organic factors playing a role in the precipitation of the psychosis. The removal of cerebrospinal fluid may cause post-lumbar puncture headache, for which many factors have been considered of etiological significance. Neurosurgeons have observed the onset of stupor and confusion following a sharp reduction in intracranial pressure due to massive loss of cerebrospinal fluid. Inasmuch as only 10 cc. of fluid were removed, there was no significant hypotension on the final manometric readings and the characteristic headache was absent; it is doubtful that the mechanical disturbance was of sufficient intensity to account for the subsequent psychosis.

There was nothing in the meager developmental history to indicate the likelihood of his post-lumbar puncture reaction, other than shyness and hints of excessive closeness to his parents. The later history revealed some indication of sexual problems, in that the patient married late in life and two marriages ended in failure. There is also the complaint of excessive sexual demands voiced by the second wife. At the time of the lumbar puncture, it was apparent that the transference was essentially a dependent one with some latent homosexual overtones, but the significance of this in terms of performing a lumbar puncture was unfortunately not considered. The "attack from the rear", along with the necessary manual manipulation of the patient's back, was interpreted by him to be an erotic, seductive attack as indicated by his comments. He referred to it as "almost pleasant". His subsequent need to portray himself as unfriendly and hostile at the time was an indication of his unwillingness to participate in the homosexual experience and his unconscious anger at being forced to do so. A further complication was the illness of the therapist which probably mobilized the patient's guilt and was a frightening confirmation of the omnipotence of his own rage. In spite of his need to defend himself against his homosexual impulses, the instinctual demand proved too great, however, and there were frequent references to a wish to be beaten by the therapist. This represented both a desire for a passive, masochistic, sexual relationship with the therapist and the retribution for this forbidden wish.

Summary

A patient who reacted to a lumbar puncture with an acute psychotic episode has been

described. It is by no means implied that the spinal tap *per se* was either the etiologic or precipitating agent. The procedure was primarily important in producing this reaction because of the total situation in which it occurred. Although the operation well might have aroused similar impulses and anxieties at any time in the patient's life and by any other physician, the fact that it was done at this particular time and by a doctor toward whom there already were conflicting emotional attitudes was the most significant factor in the production of the malignant disorder. The lumbar puncture is one of several manipulations which allow easy elaboration of homosexual fantasies. Perhaps similar episodes have been overlooked or have been diagnosed as idiopathic or unrelated intercurrent psychological illnesses by others. The failure of this patient's therapist to realize the psychological significance of a somatic procedure points up the need to be aware of the emotional impact of various diagnostic and therapeutic procedures.

BIBLIOGRAPHY

1. Brain, W. R.: *Disease of the Nervous System*, 4th Ed. London: Oxford University Press, 1951.
2. Dana, C. L.: Puncture headache. *J. A. M. A., 68:* 1017, 1917.
3. Davenport, K. M.: Post-puncture reaction. *N. Y. State J. Med., 39:* 1185, 1939.
4. Dripps, Robert D., and Vandem L. D.: Hazards of lumbar puncture. *J. A. M. A., 147:* 1118, 1951.
5. Glesne, O. G.: Lumbar puncture headaches. *Anesthesiology, 11:* 702, 1950.
6. Grinker, Roy R., and Bucy, P. C.: *Neurology*, 4th Ed. Springfield, Illinois: Charles C. Thomas, 1949.
7. Moulton, R.: Oral and dental mainfestations of anxiety. *Psychiatry, 18:* 261, 1955.
8. Pappenheim, M.: *Die Lumbalpunktion.* Wien: Ricola Verlog, 1922.
9. Redlich, F. C., Moore, B. E., and Kimbell, I.: Lumbar puncture reactions: relative importance of physiological and psychological factors. *Psychosom. Med., 8:* 386, 1946.
10. Wilson, S. A. K.: *Neurology.* Baltimore: The Williams & Wilkins Co., 1941.

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DETECTION OF PHENYLPYRUVIC OLIGOPHRENIA[1,2]

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Phenylpyruvic oligophrenia is a condition of mental deficiency which has been characterized by the appearance of phenylpyruvic acid in the urine. Folling (2) observed that this acid could be detected in the urine of these patients by the addition of a few drops of 5 per cent ferric chloride, producing a green color. While other tests have been reported which successfully detect phenylpyruvic acid in the urine (1, 3), the ferric chloride reaction has been the one commonly employed in screening mentally defective patients for this metabolite.

This communication would like to call attention to the inadequacy of employing ferric chloride alone as a diagnostic reagent.

In a survey of some 1,700 institutionalized mentally defectives, 23 patients gave a positive reaction to the ferric chloride reagent. When subsequent confirmation of the presence of phenylpyruvic acid in their urine was attempted using 2,4-dinitrophenylhydrazine, only 21 of the 23 gave a positive test. Further examination of the urine of these patients showing false positive reactions with ferric chloride were carried out. It soon became clear that the ferric chloride reaction was due to the presence of a metabolite of chlorpromazine and not to phenylpyruvic acid. Chlorpromazine itself does not react with ferric chloride to give a green color; however, a metabolite of this drug does appear in the urine which does react with ferric chloride to give a green color. This metabolite can be detected from 4 to 6 hours after the injection of a single 30-mg. dose of the drug. The recently reported metabolite chlorpromazine (4), chlorpromazine sulfoxide, did not react with the ferric chloride reagent and thus is not responsible for this reaction. Besides the test with ferric chloride the presence of the metabolite can be detected in the urine by its reaction with diazotized sulfanilic acid as follows:

To a few ml. of urine is added 5 ml. of 0.5% sulfanilic acid in 0.5 N HCl.

To this mixture is added 5 ml. of 0.5% sodium nitrite.

An immediate appearance of a purple color is noted.

Because of the increasingly prominent role chlorpromazine is playing in psychiatric medication, attention should be given to the above facts when screening mentally defectives for phenylpyruvic oligophrenia.

The author would like to express his thanks to Dr. Hill and Dr. Bogge of Fairview Home, Salem, Oregon, and to Dr. William Thompson of Morningside Hospital, Portland, Oregon, for their invaluable help in obtaining urine specimens from their patients for this study.

We wish to thank Dr. Glenn E. Ullyot of Smith, Kline & French for his generous sample of chlorpromazine sulfoxide.

BIBLIOGRAPHY

1. Delay, J., Desgrez, P., and Delbarre, F.: L'oligophrenie phenylpyruvique. *Bull. Mem. Soc. Med. Hop. Paris, 62:* 300, 1946.
2. Folling, A.: Utskillelse av fenylpyroduesyre i urinen som stofskiftesanomali forbindelse med imbesilitet. *Nord. Med. Tidskr., 8:* 154, 1934.
3. Penrose, L. S., and Quastel, J. H.: Metabolic studies in phenylketonuria. *Biochem. J., 31:* 266, 1937.
4. Sulzman, N. P., Moran, N. C., and Brodie, B. B.: Identification and pharmacological properties of a major metabolite of chlorpromazine. *Nature, 176:* 1122, 1955.

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SOCIETY PROCEEDINGS

NEW YORK ACADEMY OF MEDICINE, SECTION ON NEUROLOGY AND PSYCHIATRY AND THE NEW YORK NEUROLOGICAL SOCIETY

Joint Meeting, May 8, 1956

Joseph W. Owen, M.D., Chairman, Section on Neurology and Psychiatry, presiding

STUDY OF MOTIVES IN THE "PSYCHOPATHIC PERSONALITY". DR. JAMES R. WARE AND DR. KENNETH L. CROUNSE, New York Hospital–White Plains Division.

An analysis was made of the case studies of 40 selected patients who had been classified as psychopathic personality with or without psychosis at the New York Hospital, Westchester Division. The material in the hospital records was supplemented by follow-up information. A definite outcome was established in 32 cases. This included 10 social recoveries for a 5 year period. In the most successful cases, the therapy appeared to have been an amalgamation of active discussion of the psychopathic behavior, confrontation regarding the consequences of actions, and firm admonition. On the basis of associated symptomatology, the 40 patients were found to fall descriptively into 3 roughly equal groups. The groups each exhibited an internal consistency with regard for 1) certain aspects of the family constellation; 2) the quality of the outcome, and 3) the characteristics of the therapy most effective for that group. The first group represented the classical psychopaths without understandable motivation, and their aberrant behavior yielded only to continual management. The second group revealed a strong schizoid admixture or even a variant of schizophrenic psychosis in the follow-up period. Their families were marked by a wide physical or characterological divergence between the parents. The patients were motivated toward wholly unrealistic goals. The third group were considered to represent a variety of character disorder in which a neurotic acting-out had assumed severe proportions. This behavior was seen as an attempt to influence distorted relationships. This group had the best prognosis.

PSYCHIATRIC MANIFESTATIONS IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS. DR. JOHN F. O'CONNOR, Psychiatric Institute.

Forty cases of acute disseminated lupus erythematosus seen on the Medical Service of the Presbyterian Hospital from January, 1950 to September, 1954 were studied. Twenty-one of the cases suffered psychotic episodes. Five had severe neurotic symptomatology. Fourteen were without overt symptoms of a psychiatric nature. The group that was psychotic had a more severe form of the disease, evidenced by the length of stay in the hospital and the dosage of cortisone required. Their daily dosage was twice the amount required by the non-psychotic group. Their psychoses became apparent approximately 12 days after the institution of steroid therapy in the hospital, with a maximum of 30 days and a minimum of 2 days. Eighteen of the 21 patients who became psychotic were on steroid therapy at the time. Eleven patients who had psychoses were readmitted and started on equivalent dosages of steroids. In only 3 patients was there a recurrence of the psychosis. There was no evidence of any alternation of symptoms. The reverse seemed to be true that exacerbations of the medical illness increased the possibility of a psychosis.

Fourteen of the patients had neurological manifestations. These generally, though not always, occurred late in the disease and while on steroids. The neurological symptoms did not follow any specific pattern and it was difficult to localize the lesions. Eleven patients were autopsied and ten had pathologic changes in the central nervous system, particularly the cerebrum. Of the ten, all manifested either psychiatric or neurological symptomatology during their lives.

The prognosis for the psychotic episode was good; only one lasted more than a period of three months. Management consisted mainly of psychiatric care and gradual withdrawal of the drug as medically indicated.

DISCUSSION

DR. LAWRENCE C. KOLB: It is my wish to comment on the papers by Drs. O'Connor and Rosenkotter (which follows), as I have some acquaintance with their material. Dr. O'Connor has done us a service in his study on lupus erythematosus, particularly in relation to one point. For some time now it has been the general opinion that individuals who have a history of previous psychotic breakdown or emotional disturbance should not be treated with the steroids. This opinion derived largely from the studies of persons who have developed psychotic breaks or other disturbed states in the course of treatment of rheumatoid arthritis. Dr. O'Connor's findings that patients who have had one psychotic breakdown in the course of steroid treatment of lupus erythematosus did not usually become disturbed with the institution of a second course of steroid treatment is therefore a fact of considerable interest. Frequently I have recommended that patients with rheumatoid arthritis who have had a psychotic disturbance in the past on treatment with steroids should not again be treated. However, the propriety of such a recommendation now is open to question.

DR. MARVIN STERN: I would like to discuss Dr. O'Connor's paper and compare it with some of the Bellevue material. This is an interesting study, and contributes a significant series to a select few large series of patients, who have been studied in any one hospital. As the series have been collected, the percentage of psychoses reported has been continually increasing, and this series represents the highest percentage of psychoses I know of, except in the Bellevue series. There is reason for the latter, since the primary reason for patients admission is often psychiatric, and these are computed with the general hospital admissions. In terms of the general categories described, they resemble our material except that we see also manic episodes.

In terms of the relationship to cortisone treatment, in all the series now, as the dosage and duration of treatment increase, the psychotic reactions rise. I think it is important to point out one cannot correlate precisely statistically the incidence of psychoses with the level of cortisone in a series of patients, or even in the same patient at different times, because those patients who have become psychotic at one level of cortisone return to treatment and are not noted to be psychotic at another level, even higher than the original.

In one-half of our patients who were seriously involved psychiatrically, we felt the brain damage was related to the lupus activity rather than to the cortisone. In some patients already psychotic they received as much as 600 mg. daily for three to four months with eventual recovery, so one should not be frightened by the psychosis in relation to cortisone. Our feeling is that the psychoses tend to remit, whether with the reduction of cortisone or of lupus activity.

I was overwhelmed by the high incidence at autopsy of brain pathology, and I think this contradicts some of the evidence previously described. We have reviewed the records of closely observed patients who had extensive lupus activity, and who had undoubted brain and systemic involvement, and at postmortem we saw only few traces of lupus activity.

DR. JOHN F. O'CONNOR: The high incidence of psychoses in the series might be due to the extensive follow-ups we had, and if we could follow all these patients we might even have a higher incidence. The autopsy correlation was extremely high and with such a small group may well be coincidental, but pathologic findings in the central nervous system are not uncommon in the literature.

DR. RICHARD M. BRICKNER: Can you tell us some more about the neurological picture in these patients during life?

DR. O'CONNOR: This was out of my field, but as I said, the neurological picture was variable. Lesions were difficult to localize. One patient had slight hearing loss. Two of the patients presented themselves initially to a neurologist for examination:—one as a quadriplegic. The lupus symptomatology was very much overshadowed by the neurological symptomatology. There was no specific symptomatology and it was always accompanied by evidence on neurological examination of malfunction in the central nervous system. Interestingly enough, one of the patients presented as a schizophrenic patient and then manifested symptoms of lupus erythematosus.

ACUTE RELIGIOUS PSYCHOTIC STATE. DR. LUTZ ROSENKOTTER, Psychiatric Institute.

A 20 year old college student, son of a minister, was admitted to New York State Psychiatric Institute after having developed a psychosis of one week's duration with catatonic and paranoid manifestations and with religious content. This psychosis manifested itself when he had entered a chapel to pray for religious awakening. In doing so he followed literally the suggestion of his father with whom he had had arguments of a religious and philosophical nature.

Ministry had been practiced in the father's family for three, and in the mother's family for four generations, and it was considered a family tradition. The patient's relationship with his mother was marked by lack of warmth and affection; he therefore had identified entirely with his father at the price of submission under his father's dominance and his rigid religious ideas. He had always identified his father with God; rejection of his father's dogma meant separation from the only source of approval and security, and at the same time, blasphemy, thus causing unbearable anxiety. When this was pointed out to the patient, he was able to give up his psychotic "retreat from reason" and to work successfully in psychotherapy. He was discharged three months after admission, having received only psychotherapy, and is now continuing his college education without recurrence of psychotic symptoms.

DISCUSSION

DR. LAWRENCE C. KOLB: Dr. Rosenkotter's case presentation provides the most manifest account of the connection of a religious conversion as the facade for a psychotic reaction. In his adolescent conflict with his father the patient attempted to revolt against the father's authority which dictated his choice of profession in the ministry. Unable to face his emerging hostility and accept a loss of his dependent relationship on the father, he selected as a solution the very course suggested by the father prior to his return to college, that is, the conviction of a religious conversion. Here we see that the specificity of choice of symptoms was determined through the parental suggestion, in short, this was the only course of action open to the young man on the basis of his family experiences.

Dr. Rosenkotter is to be congratulated on his able and sensitive management of this particular patient. He provided the initial interpretation which led to the therapeutic opening up of the case and its eventual successful outcome.

DR. GOTTHARD BOOTH: I have been consulting psychiatrist of General Theological Seminary in New York for the past 20 years and have noticed that psychotherapy with people who came from a theological background is generally much easier than it is with the average case. It seems to me that one of the reasons for this is that people who come from a tradition or an educational background in the sense that they believe in the reality of the soul more than the case of the average more worldly person, are much more responsive to psychotherapy. One can explain it in this way, that in psychotherapy of the average more worldly person we have to spend most of our time in trying to convince the patient of the reality of the soul, whereas where

people are already convinced and believe in its reality, the usual time-consuming preparation for therapy has been accomplished beforehand. I have seen in my counseling work with theological students over and over again cases I would expect to take a great deal of time for psychotherepy who responded much more quickly than nontheological patients.

DR. ROSENKOTTER: I agree with Dr. Booth that this patient responded more readily to psychotherapy because of his spiritual orientation. Religious matters were occasionally discussed in the course of treatment, and I tried to convince the patient that his father's rigid attitudes are not necessarily rooted in Christian religion.

RUPTURED THORACIC INTERVERTEBRAL DISK CAUSING CORD COMPRESSION IN A CHILD. DR. FREMONT C. PECK, JR., New York Hospital.

The case of a 12 year old boy with a calcified nucleus pulposus at T-6 which had ruptured into the spinal canal, causing signs of cord compression, is reported. Tomograms of the spine and pantopaque myelography clearly defined the lesion. The protruded calcified nucleus was removed at laminectomy and the cord signs relieved. A brief review of the pertinent literature and a discussion of the relationship between the calcification of intervetebral disks in childhood and peritendinitis calcarae are included.

HEAT STUDIES ON SOME NEUROLOGICAL DISEASES. DR. DEWEY A. NELSON AND DR. WILLIAM H. JEFFREYS, Bellevue Hospital, Neurological Second Division.

This study was undertaken to examine a number of diseases of the nervous system after immersion of the patients in hot water, to observe for changes in the vital signs and in certain areas of the neurological examination. Sixteen normals and 84 patients with various diseases of the nervous system, including 12 patients with multiple sclerosis, were studied.

The patients were first examined as regards vital signs, visual acuity, strength of the extremities, deep tendon reflexes, plantar responses, dysarthria, nystagmus, range of extra-ocular movements, pupillary size, and mental state. The patients were then immersed in an ordinary bath tube to the nipple-line in water of 104°F. The water temperature was then elevated to 110°F. and the patients were left in the water for 20 to 30 minutes. During this time they were examined in each category 4 to 5 times.

Of the 16 normals, 13 had no neurological change (except mental changes), two developed tetany and one a decrease in grip.

Of the 84 patients with neurological disease, 33 had no change except mental changes and 51 demonstrated alteration in the neurological examination. Seven patients had a decrease in visual acuity. Of these, five had the diagnosis of multiple sclerosis. Four patients showed gross visual field changes and two of these were confirmed by immersing the feet and lower legs alone. No patients with multiple sclerosis were examined for visual field changes because of nystagmus interference. In 33 patients the development of, or increase in, nystagmus was noted. In this group there were 9 patients with multiple sclerosis, 22 patients with diffuse dysfunction of the central nervous system, and only 2 patients with no disease of the central nervous system. Nine patients, all with the diagnosis of multiple sclerosis, developed a medial longitudinal fasciculus syndrome. Six of these patients either had or developed prior to the onset of the longitudinal fasciculus syndrome more nystagmus in the abducting eye in lateral gaze. Ten patients had reflex changes, all of which involved the ankle jerks only. Sixteen patients with neurological disease showed a change in grip. Four patients had a change in plantar responses, two to normal and two to abnormal. Two patients had seizures, three ptosis of the lids, two individual extra-ocular muscular palsies, and one patient became unresponsive for a short period of time.

Vital sign and mental changes observed in this study conformed to those previously described; there were no significant differences between the normal, change and no-change group.

Individually, the patients in the multiple sclerosis group were most profoundly affected by this experimental situation, but they represented only 25 per cent of the patients who showed neurological changes.

DISCUSSION

DR. RICHARD M. BRICKNER: I should like to ask Dr. Nelson and Dr. Jeffreys if they ever saw any permanent residual effects.

DR. JEFFREYS: I am sorry I did not mention that. Most of the changes we saw, and some were quite dramatic, would resolve within 5 to 10 minutes of withdrawing the patients from the hot water. One patient I mentioned who went into coma came out of her unresponsive state in four minutes when we put cold water in the bath. All these changes seem to be rapidly reversible.

DR. BRICKNER: I ask this question because I have seen some which were lasting.

Also there are experimental situations in which you can offset the neural reactions while the heat (dry) is still on. I have found with a few that vasodilating drugs will sometimes do this in cases where heat impairs visual acuity or induces nystagmus.

Can you make a little clearer what you mean by the statement that multiple sclerosis patients are more profoundly affected than others, and just what you mean by the humoral theory suggestion?

DR. JEFFREYS: As far as the multiple sclerosis patients being more profoundly affected, this was the only category in which we found more than two neurologic changes; many of these patients developed not only the changes which we felt to be those of the medial longitudinal fasciculus syndrome, as you saw in five cases, but visual acuity changes, and also reflex and drift changes. They seemed to have a more widespread manifestation of intolerance to heat.

As far as the humoral theory is concerned, some of these patients developed changes prior to any significant change in vital signs. As you recall the work by Guthrie, he was able to reproduce these changes by merely immersing one arm. He was able to get around these changes by putting on an arterial tourniquet. We feel that since these changes are so rapid in onset, and so quickly reversible, and may be produced by immersing a portion of the body that the most reasonable explanation was the production of some substance in the area that was heated.

DR. THOMAS K. DAVIS: There is only one point I would make in regard to the multiple sclerosis report. One cannot be surprised by what has been reported here tonight. In the treatment of multiple sclerosis there has been a period when many people believed in using fever-producing typhoid vaccine. I do not know whether this is still being done. I never have approved of doing so, and only want to refer briefly to the teaching of Dana, one of this Society's really great ancestors. He taught that nothing should be done in the treatment of multiple sclerosis that was on the strenuous side. I do not believe that patients with multiple sclerosis can stand strenuous treatment, or strenuosity in any form.

DR. MORTON NATHANSON: Heat seems to be just one of the conditions that will produce changes in people who have disease of the nervous system. It even may produce measurable neurologic changes in the normal individual. It has been shown that heat, severe cold, barbiturates, alcohol and even high doses of cortisone commonly produce changes in the nervous system. Some of the changes are predictable; others are not. For example, barbiturates may stop certain types of nystagmus with subsequent improvement in vision. In a given patient it will stop a nystagmus on direct forward gaze and enhance or increase nystagmus on lateral gaze. Then we may observe a patient with multiple sclerosis who is depressed and under the influence of barbiturates becomes euphoric. I emphasize these points to warn against drawing conclusions as to the etiology of multiple sclerosis based on a change in the conditions of testing.

As far as what Dr. Davis said about the use of typhoid vaccine, there were times when high temperatures were said to produce, or to be a contributing factor in producing a remission in patients with multiple sclerosis. Here was an example where temperature or heat produced a remission of objective signs.

DR. IRVING J. SANDS: Did you have any EEGs done on these patients?

DR. JEFFREYS: Yes.

DR. DEWEY A. NELSON: I should like to emphasize one thing which Dr. Jeffreys summarized and mention one practical point. The first of these is that neurological changes can be quite transitory. We have seen medical people who have done few neurological examinations seem surprised that they do not see at the bedside today what they were sure they observed yesterday morning. Perhaps neurological changes occur frequently when the patient is febrile, and I think we should all observe for this. For example, Dr. Jeffreys, Dr. Wainerdi and I one afternoon at the Hospital for Special Surgery observed the disappearance of a medial longitudinal fasciculus syndrome on defervescence with residual dissociated nystagmus and this occurred over a 90 minute period. This patient had multiple sclerosis.

The second point is that I think we should warn patients with multiple sclerosis against taking unsupervised hot baths due to the very real danger of their slipping beneath the water.

TRANSIENT DISPLACEMENT OF THE PINEAL BODY. DR. BERNARD COHEN AND DR. SUN KYOO SONG, Third Division Neurology Service, New York University-Bellevue Medical Center.

The spontaneous return towards the mid-position of a previously shifted calcified pineal body was observed in five cases of intra-cranial disorder. In four cases the displacement was lateral, in one case posterior. The etiology in three cases was probably trauma and vascular disease in the other two. In each case the shift was a significant one, from 4 to 7 mm. Initially, the x-ray findings and the clinical picture suggested the presence of a subdural hematoma or a brain tumor. Despite the pineal shift all of the patients showed clinical improvement and were treated conservatively. Pneumoencephalography was done in all patients and carotid angiography in two. No surgical procedures were performed. All of the patients had a relatively complete recovery. Edema and/or intra-cerebral bleeding were assumed to be causing the pineal shift. The conclusion drawn was that even in the presence of obvious pineal displacement the clinical signs must be evaluated carefully. Patients with expanding tumors or subdural hematomas rarely, if ever, show sustained improvement. If this improvement occurs, there is indication for continued conservative management. Thus, patients with edema of the brain or intra-cerebral bleeding can be spared unnecessary burr holes, ventriculography, or craniotomy.

DISCUSSION

DR. MORTON NATHANSON: I believe it is important to emphasize the management of patients with shift of the pineal body. At one time before the use of angiography, when a patient was admitted to the hospital with a history of head trauma and a shift of the pineal was found, burr holes were done immediately. We found that some of the patients did not have a subdural hematoma, despite the shift of the pineal. However, more recently angiograms are performed where there is a shift of the pineal, and although there may be bowing of the anterior cerebral artery, there often is no evidence of subdural hematoma. We therefore observe the patient, and as has been shown tonight, the pineal may shift back to its normal position. More recently in the reported cases of occlusion of the internal carotid artery, it has been not uncommon to find a shift of the pineal. I believe we should bear in mind that a shift in the pineal body *per se* without any clinical evidence of a downward course should be handled conservatively.

DR. GEORGE H. HYSLOP: This is not a discussion: it is a reminder of what the presenter of the last paper perhaps overlooked. He spoke of cerebral hemorrhages as responsible for some of the pineal shift. Cerebral hemorrhages practically all lead to death. We do not have a cerebral hemorrhage produce a transient pineal shift and a relatively benign clinical picture. We are in these cases dealing with a thrombotic reaction and surrounding edema, a long recognized condition.

DR. BERNARD COHEN: We have seen many patients with vascular accidents, grossly bloody spinal fluid, and localizing cerebral signs in whom there was apparent hemorrhagic involvement of the cerebrum. Many of these patients have later recovered. Since in the cases we presented there was no direct evidence of the pathologic process, we feel that the possibility of extravasation of blood into the substance of one hemisphere must be considered along with cerebral edema as a possible physiological mechanism causing a transient pineal body shift.

BOOK REVIEWS

Delay, Jean: ASPECTS DE LA PSYCHIATRIE MODERNE. Paris: Presses Universitaires de France, Pp. 115, 1956.

This small paper-covered book is composed of three essays on present day psychiatry by the Professor of Psychiatry of the Faculty of Medicine of Paris. The first is an inaugural lecture delivered for medical students in which historical and modern achievements of psychiatry and disorders of the brain are reviewed in terms of theory and practice.

The second essay was presented as the opening lecture of the first world psychiatric congress held at the Sorbonne in September, 1950. The discussion here covers the modern psychiatric researches and ideas that were most prominent in the laboratories and clinics of the civilized countries.

The third essay is entitled "Néurose et Création", the presidential address given by the author on the occasion of the 52nd congress of French neurologists and psychiatrists held at Liege in July, 1954. In this presentation one finds an interesting account of the possible influence of nervous disorders suffered by outstanding creators, on creative thought and on their contributions to philosophy, literature and science. The three essays are well worth reading for students of medicine and psychology.

Cauldwell, David O.: TRANSVESTISM. New York: Sexology Corporation, Pp. 128, Illustrated, 1956, $3.00.

This book, apparently the first to be devoted entirely to transvestism is presented in two parts. The first is entitled "The Origin and Nature of Transvestism" and consists of seven articles by as many different contributors. The second part is composed of eleven "Autobiographic Case Histories" written by male transvestites who have signed their feminine names.

Although the term "transvestism" is applied to a deviation in which an individual of one sex dresses in the clothes of the other sex, the text in this book deals principally with the male expressions of this perversion, and has obviously been written for the laity rather than for the psychiatrist, who by the way, will find many interesting phenomena to provoke thought.

Many transvestites emphasize that they are normal otherwise and sometimes argue that even this is a unique, normal desire but it is well known to psychiatrists that such trends are rationalized in various ways by the subject, with often little distinction made between homo and heterosexuality. One should avoid taking their own statements always at face value since some of these eonists understate or inaccurately report their behavior and feelings to disguise, in part, the actual situation. The impulse is certainly widespread with the majority of the instances kept secret by the practitioners who realize that it is a perversion of the sex life.

If the term "homosexuality" is reserved for the overt act of mutual genital stimulation between persons of the same sex, and the term "homoerotocism" for masked or implicit homosexuality, many issues which the authors of this book discuss and on which pronouncements are made will become clearer and less contradictory.

One feels that if more were known about the transvestites reporting in this book that the findings and impressions gained by psychoanalysts who have dealt with similar cases would become quite obvious. Psychoanalysts have found the edipus situation here pronounced, beginning early in life and having been never resolved, there is a strong identification with the mother and/or sister. They are all strongly skin erotic, expressing exquisite pleasure at the feel of feminine garments on their bodies and often so sexually stimulated by their feminine attire that they masturbate. The transvestite satisfies himself with the appearance of belonging to the opposite sex and are in rivalry with the opposite sex. A psychoanalyst would surely find the wives of the married male transvestites, who know and even promote this psychopathological impulse in their mates, very interesting psychological studies.

Although there are many manifestations and

variations in transvestism the base line is in the realm of homosexuality in the passive role with narcissization of the whole body as in females. The proof of this is the ease in which they are seduced into overt homosexual practices. Usually one seduction or experience releases the force.

The role of parents particularly mothers in creating this condition in little boys by dressing them in girls' clothing for forms of punishment or perhaps more often to express the wish for a girl child, is strongly and rightly emphasized by the authors of this book. But there must be some other factor, acting as a predisposition in these children since many boys have been dressed as girls for punishment, for ridicule, and for the fashion in the 1880's and the vast majority of these have had no tendency to prolong the situation having gained no secret pleasure from it.

There is a great deal of valuable information collected in this book and the material is interestingly presented and particularly well illustrated.

Grinker, Roy R. (Ed.), TOWARD A UNIFIED THEORY OF BEHAVIOR. With assistance of Helen MacGill Hughes. (New York, New York: Basic Books, Inc., pp. xvi + 375. 1956. $6.50).

The idea of a unified and universal science, or more exactly, the search for a limited number of basic concepts and principles which would form part of any scientific system designed to explain changes in reality perceived through the senses, received a new and powerful impetus from modern non-Aristotelian logic about fifty years ago. A new science (or theory) of science developed which promises to be more fruitful than any previous attempts at a universal science. The limits of specialization have now been largely recognized and a need for interdisciplinary cooperation has arisen. This cooperation results only too frequently in an accumulation of diverse data which cannot be intelligibly integrated. A scheme is needed to make the inter-disciplinary contacts scientifically profitable. Grinker, the chairman of the conferences reported in this volume, describes the main purpose as: "developing concepts from each field and attempting to derive from them abstractions applicable to all fields". The conferees were biologists, psychologists, psychiatrists and social scientists, experts in the sciences of human behavior. Physicists, chemists, and other physical scientists were not present. The conferees faced a most difficult problem even in their quest of concepts applicable only to the four sciences dealing with human behavior. To be scientifically fruitful, such concepts must express some very basic functions of the mind and refer to some very basic aspects of perceptual reality which can be identified easily and reliably.

This book covers the first four of the conferences and therefore does not contain the final conclusions. It is very instructive to follow the discussions and to see how frequent was the temptation to ascribe perceptual reality to what are but classificatory ideas or to consider as an entity something that is but an integral part of a pattern. In view of the difficulty of the problem it is understandable that the discussion usually centered around specific methodological issues of specific sciences, that it was mostly descriptive and educational in the sense that the specialist on a particular problem gave instruction to the non-specialists, prompted by the latters' probing questions. As in other individual or group attempts of the same kind, most of the discussion consists of restating the problem rather than of coming to grips with it. The simple concepts that have been suggested as universal in application have for the most part been borrowed from physics. They have the advantage of clarity and simplicity but do not carry explanation very far in fields more complex than elementary physics. In the attempts at formulating a unified theory three currents can be distinguished, not always easily. One aims at essentially letting the particular sciences develop independently of one another but trying to integrate their diverse results on the assumption that each studies a different aspect of the human being. Thus laboratory tests, psychiatric clinical examinations, psychological tests, social data, can be combined to gain a better understanding of the individual. Another current aims at creating concepts and principles which would accomplish more than an addition of results obtained by different scientific methods; it hopes to provide a procedure that would synthesize the different methods, making of them parts of a highly integrated inductive

and deductive pattern. The third current aims to find the concepts and principles basic to all empirical sciences regardless of their specific methods and subject-matter. These currents can be reconciled but their goals are not identical. If these goals were more clearly defined and the differences kept in mind, the theoretical discussions might become easier and more fruitful. Although the nineteen conferees fell short of their main goal (which is a task for generations), they are recognized leaders in their field and therefore the transactions contain very instructive and highly interesting material. Theorists and those actively engaged in research in the behavioral sciences will enjoy this volume and benefit from it most.

ZYGMUNT A. PIOTROWSKI

Cooper, Irving S.: The Neurosurgical Alleviation of Parkinsonism. Springfield, Ill.: Chas. C Thomas, Pp. 104 with 68 illustrations, 1956, $8.50.

In the preface of this timely book, the author, an experienced neurosurgeon and teacher, outlines six principal objectives which are presented and elaborated in the text, namely 1) evidence that certain neurosurgical procedures are applicable and indicated in many severe cases of parkinsonism, 2) synopsis of both the earlier and contemporary neurosurgical techniques for the therapy of this disorder, 3) detailed descriptions of the operative techniques developed in the author's clinics, 4) a summary of the results obtained in his personal series of over 125 cases, 5) to inform physicians of the therapeutic possibilities in this neurosurgical approach to Parkinsonism, by means of adequte case histories and 6) an attempt to point out new possible neurosurgical approaches to the hyperkinetic disorders.

There are eight chapters in the monograph, the first of which is devoted to a brief but adquate outlining of the problem and description of the symptoms of Parkinsonism. The second chapter presents a review of the various surgical approaches to the treatment of the disorder, the operations being excellently illustrated by diagramatic representations. Chapter three gives the anatomy, physiology, pathology of the globus pallidus and its role in the syndrome. Chapter four contains the description of surgical occlusion of the anterior choroidal artery, an operation developed by the author. Here is the detailed account of the preoperative procedures, the surgical technique, post operative care and the evaluation of results in terms of each of the symptoms. In chapter five one finds 15 representative case reports in detail, liberally illustrated and pointing out the important issues.

Chapter six presents a chemopallidectomy procedure also developed by the author and lucidly described in order from the preoperation preparation of the patient to the final results. In this therapy procaine is injected into the area to alleviate contralateral tremor and rigidity. This is followed by the injection of absolute alcohol to produce a destructive or neurolytic lesion. This operation has been performed 70 times. "In 85% tremor and rigidity was satisfactorily alleviated at the initial operation." Chapter seven is composed of ten informative case reports on the results of chemopallidectomy and finally chapter eight gives the summary and conclusions, among which one finds this statement which is important to all physicians: "Surgical techniques are available, which if meticulously executed in the proper cases can produce good results in 70% of Parkinsonian patients chosen for operation."

The text throughout has been organized logically in terms of indications, surgical techniques, complications, results, follow-up data, etc., in their proper places to be of value not only to neurosurgeons and neurologists, but also to internists, general practitioners and students of medicine. The illustrations are remarkably clear. The page trim size is 8½ x 11 inches with the two column type on Cumberland Gloss paper, and strongly bound thus producing an attractive, durable book, as well as a fine contribution to neurosurgery.

BOOKS RECEIVED FOR REVIEW

THE JOURNAL OF NERVOUS AND MENTAL DISEASE

May, 1956

1. American Psychiatric Association. Psychiatric Research Report 3. Conference held at Duke University School of Medicine, Durham, North Carolina, November 19–20, 1954. American Psychiatric Association, Washington, D. C.
2. Centennial Commission, Saint Elizabeths Hospital. Centennial Papers, Saint Elizabeths Hospital 1855–1955. Waverly Press, Inc., Baltimore, Md.
3. FAUST, CLEMENS. Das Klinische Bild der Dauerfolgen Nach Hirnverletzung. Georg Thieme Verlag, Stuttgart, Germany.
4. FREDERICK, PORTIA M. AND TOWNER, CAROL. The Office Assistant in Medical or Dental Practice. W. B. Saunders Co., Philadelphia, Pa.
5. GESELL, ARNOLD, ILG, FRANCES L. AND AMES, LOUISE B. Youth: The Years From Ten to Sixteen. Harper and Bros., New York, N. Y.
6. GRINKER, ROY R. (Ed.) Toward A Unified Theory of Human Behavior. Basic Books, Inc., New York, N. Y.
7. GRUHLE, HANS W. Verstehende Psychologie. Georg Thieme Verlag, Stuttgart, Germany.
8. JAKOBSON, ROMAN AND HALLE, MORRIS. Fundamentals of Language. Mouton & Co., The Hague, Holland.
9. KRETSCHMER, ERNST. Medizinische Psychologie. Georg Thieme Verlag, Stuttgart, Germany.
10. REHWALD, E. Das Hirntrauma. George Thieme Verlag, Stuttgart, Germany.
11. SARTRE, JEAN-PAUL. Being and Nothingness: An Essay on Phenomenological Ontology. Philosophical Library, Inc., New York, N. Y.
12. SCHWARTZ, MORRIS S. AND SHOCKLEY, EMMY L. The Nurse and the Mental Patient. Russell Sage Foundation, New York, N. Y.
13. STEVENSON, GEORGE S. Mental Health Planning for Social Action. McGraw-Hill Book Co., Inc., New York, N. Y.
14. TRUSSELL, RAY E. Hunterdon Medical Center: The Story of One Approach to Rural Medical Care. Harvard University Press, Cambridge, Mass.

NOTES AND NEWS

THE FORD FOUNDATION PROGRAM

Twenty-one research centers have been selected to receive grants under The Ford Foundation's program for strengthening and extending research in mental health, it was announced today.

The grants, totaling $6,826,850, were allocated from a $15,000,000 appropriation announced by the Foundation in May 1955. They will be used for research over the next five years.

The 21 projects approved for support by the Foundation were selected from a total of 231 applications. The Foundation action covers only applications from the United States. Approval of European applications will be announced. later.

The grants cover a wide range of investigations in the field of mental heath—by subject, by approach, by type of institution and by geography. Research activities for which the Foundation authorized grants are concerned with personality dynamics and development; biological, physiological and somatic problems; social and community aspects of mental health; children's disorders; and studies in therapy.

To assist in the development of the mental health program, The Ford Foundation's behavioral sciences staff set up an advisory group consisting of Dr. John Romano, University of Rochester; Dr. Seymour Kety, National Institutes of Mental Health and Neurological Diseases and Blindness; Dr. Merton Gill, San Francisco Psychoanalytic Institute; David Shakow, National Institute of Mental Health; Ernest Hilgard, Stanford University; and John C. Eberhart, the Commonwealth Fund.

Investigagors in the approved projects represent a broad range of disciplines bearing on mental illness, among them anatomy, physiology, pharmacology, neurochemistry, psychiatry and psychology.

In its original announcement of the program, the Foundation stated that despite the sizable need for trained personnel in the therapeutic aspects of mental illness, it felt its greatest service at this time would come through emphasis on research. Long-range goals of the Foundation-supported research include determining the causes of mental illness and developing and testing effective methods of treatment and prevention.

THE ACADEMY OF PSYCHOANALYSIS

At an organizational meeting in Chicago, on April 29, 1956, the Academy of Psychoanalysis was founded for the following purposes as specified in its Constitution:

The aims of the Academy are:

(a) To develop communication among psychoanalysts and their colleagues in other disciplines in science and in the humanities;

(b) To constitute a forum for free inquiry into the phenomena of individual motivation and social behavior;

(c) To encourage and support research in psychoanalysis;

(d) To foster the acceptance of psychoanalysis and its integration in universities;

(e) To advance the interests of psychoanalysis in all other respects.

The officers elected at the first meeting of the Academy are: Janet Mackenzie Rioch, M.D., New York, N. Y., President; Jules Masserman, M.D., Chicago, Ill., President-Elect; Frances S. Arkin, M.D., New York, Secretary and Leon Salzman, M.D., Washington, D. C., Treasurer.

Physicians, who are graduate psychoanalysts, may qualify as Fellows. Physicians and others in related fields of behavior study may apply for acceptance as Scientific Associates. Meetings will occur semi-annually. Communications to The Academy of Psychoanalysis may be addressed to the Secretary or to any of the other officers.

SEVENTH ANNUAL NORTH SHORE HEALTH RESORT LECTURE SERIES
THE MEDICAL PRACTITIONER'S CONTRIBUTIONS TOWARD HEALTHY EMOTIONAL DEVELOPMENT

Wednesdays	8:00 P.M.	225 Sheridan Road, Winnetka, Illinois
Oct. 3, 1956	THE UNIQUE POSITION OF THE PHYSICIAN IN OUR SOCIETY	DANIEL BLAIN, M.D., Medical Director, American Psychiatric Ass'n., Clin. Prof. of Psychiatry, Georgetown Univ.; Consultant, Vet. Adm.; Member, Expert Committee on Mental Health of the W. H. O.
Nov. 7, 1956	THE PSYCHOLOGICAL PREPARATION OF THE INDIVIDUAL FOR MEDICAL AND SURGICAL CARE	MILTON ROSENBAUM, M.D., Professor and Chairman, Department of Psychiatry, Albert Einstein Col. of Med.; Dir. Psych. Services, Bronx Municipal Hosp. Center; Chair. Com. on Medical Education, A. P. A.
Dec. 5, 1956	PROBLEMS OF EARLY DEVELOPMENT	LAURETTA BENDER, M.D., Prof. Clin. Psychiatry, New York Col. of Med.; Principal Research Scientist in Child Psychiatry, Dept. of Mental Hygiene, State of New York.
Jan. 9, 1957 (Second Wed.)	PROBLEMS OF THE SCHOOL CHILD	RALPH D. RABINOVITCH, M.D., Director, Hawthorne Center, Consultant in Child Psychiatry, State Department of Mental Health, Northville, Michigan.
Feb. 6, 1957	PROBLEMS OF TEENAGERS	FREDERICK H. ALLEN, M.D., Director, Philadelphia Child Guidance Center; Clin. Prof. Psych., U. of Penn.; Pres. Amer. Acad. of Child Psych.; Past Pres., Inter. Ass'n. of Child Psychiatry.
Mar. 6, 1957	PROBLEMS OF ADULTHOOD	C. KNIGHT ALDRICH, M.D., Professor and Chairman, Department of Psychiatry, University of Chicago School of Medicine.
Apr. 3, 1957	PREMARITAL COUNSELING	EMILY H. MUDD, Ph.D., Director, Marriage Council of Philadelphia; Ass't. Prof. Family Study, Dept. of Psych., School of Med., U. of Penn.
May 8, 1957 (Second Wed.)	MARRIAGE COUNSELING	ROBERT W. LAIDLAW, M.D., Chief, Div. of Psychiatry, The Roosevelt Hosp., Past Pres. Amer. Acad. of Marriage Counselors.
June 5, 1957	PROBLEMS OF GRANDPARENTS	ERICH LINDEMANN, M.D., Professor of Psychiatry, Harvard Medical School, Psychiatrist-in-Chief, Mass. Gen. Hosp.; Medical Director, Human Relations Service, Inc.

ESTABLISHMENT OF HOSPITAL CENTERS

The first of two day hospital centers to be organized by the New York State Department of Mental Hygiene will be opened at Hudson River State Hospital, Poughkeepsie, on July 2. The day hospital is one of the Mental Hygiene Department's pilot projects to determine the value of psychiatric and supportive therapy for suitable mental patients in a hospital setting during the daytime hours. Patients at the new center will receive psychiatric care on a voluntary outpatient basis, during the day, remaining for the rest of the time at their homes or continuing insofar as possible with their family and community activities.

The second day hospital is expected to open in the near future in connection with the Brooklyn Aftercare Clinic, 105 Schermerhorn St., Dr. Hoch revealed.

The Hudson River day center will occupy one ground floor wing of the new Cheney Memorial Building. This wing has a separate front entrance and forms a self-contained unit entirely independent from the hospital and the rest of the Cheney building. It consists of sixteen rooms with ample physical facilities for all types of therapies, offices, and reception services.

The treatment area will consist of a treatment unit with ten beds, an occupational therapy studio and a rehabilitation area including a library and recreation room. Dining facilities also will be available.

The day hospital has been designed for the care of adults eighteen years or older and will operate on a five-day a week schedule from 8:30 a.m. to 4 p.m. All of the standard psychiatric and somatic therapies, including drugs, electric shock, and insulin will be given.

Patients will be referred to the day hospital primarily by community physicians and social agencies and on a volunteer basis, according to Commissioner Hoch. Cases also may be accepted after provisional release from the state hospital to complete the patients' course of treatment or for social and vocational readaptation as a means of helping to "tide them over" in making an adjustment from the hospital to the community.

A staff of fourteen will administer the day hospital, headed by Dr. O. Arnold Kilpatrick, director of the Hudson River State Hospital. Dr. Marian Axel will be the psychiatrist in charge of the center. The staff will also include a psychiatrist, two nurses, a social worker, occupational therapist, recreational worker, two male and four female psychiatric aides, and a secretary-receptionist.

DR. HOCH NAMES TWO DMH PSYCHIATRIC CONSULTANTS

Two New York City psychiatrists, Dr. M. Ralph Kaufman, chief of psychiatry at Mount Sinai Hospital, and Dr. John Millet, a private practitioner affiliated with Presbyterian Hospital, have been named as consultants to the New York State Department of Mental Hygiene by Dr. Paul H. Hoch, Commissioner of Mental Hygiene.

Both men were charter members of the State Mental Hygiene Council and recently completed their terms of office after serving five years.

Dr. Hoch declared that the appointment of the two psychiatrists to the honorary posts was made in recognition of the outstanding services they have performed for the department especially during their tenure on the Mental Hygiene Council.

APA ENDORSES NEW NYS LAW CERTIFYING PSYCHOLOGISTS

New York State's new law requiring certification of psychologist was endorsed last month by the Committee on Relations with Psychology of the American Psychiatric Association.

The New York law, which was enacted by the 1956 Legislature and becomes effective July 1, 1957, fully meets the criteria set up by the association, the committee reported, and is fully consistent with the policies adopted by the American Psychological Association.

Pointing out that this is the first state law to be enacted with the full approval and support of national and local groups of the two professions, the committee expressed the hope "that this is a milestone, marking an end to the difficulties which have been experienced in many states—and now surmounted in New York State; and that it will prove to be the basis for interprofessional agreement in other states."

TWO STATE HOSPITALS OPEN PILOT GERIATRIC UNITS

Two of the three experimental geriatric rehabilitation units planned for the intensified care of older mental patients in New York State went into operation last month.

The two centers are located at Central Islip and Buffalo state hospitals. The third is expected to open at Pilgrim State Hospital in the near future.

The geriatric units are being established as an experimental project under the Mental Hygiene Department's new nine-point program of intensified treatment and research. The objective, according to Dr. Paul Hoch, Commis-

sioner of Mental Hygiene, will be to find out how many geriatric patients can be sufficiently rehabilitated by intensive medical and psychiatric care to be returned to the community.

Hospital teams, including psychiatrists, nurses, social workers, occupational, recreational and physical therapists, are being set up to administer the program. A number of positions already have been created and filled at both the Buffalo and Central Islip centers.

DR. STORRS TO RETIRE AFTER 44 YEARS' SERVICE WITH DMH

Dr. Harry C. Storrs, director of Letchworth Village for the past 19 years, will retire on June 30 after 44 years' service in the Department of Mental Hygiene. For 26 of those years, he has been in charge of a state school, having been superintendent at Wassaic for seven years before he was appointed head of Letchworth Village in 1937.

NEW OFFICERS OF MASSACHUSETTS SOCIETY FOR RESEARCH IN PSYCHIATRY

The following are the newly elected officers of the Massachusetts Society for Research in Psychiatry for the year 1956/57:

President: MAX RINKEL, M.D.
Vice-President: PHILIP SOLOMON, M.D.
Secretary-Treasurer: PETER H. KNAPP, M.D.
Chairmen of Committees:
Program Committee: STANLEY KANTER, M.D.
Committee on Constitution: MYER ASEKOFF, M.D.

The schedule of the meetings of this Society is as follows:
Friday, October 19, 1956 (First Meeting)
Friday, December 14, 1956 (Second Meeting)
February, 1957 (Third Meeting)
April, 1957 (Fourth Meeting)

POSTGRADUATE COURSE IN NEUROLOGY AND PSYCHIATRY

The Tenth Connecticut Postgraduate Seminar in Psychiatry and Neurology will begin its courses of lectures on September 19th and will continue through May 6, 1957.

There are no fees for any of the courses.

Copies of the program may be obtained from the Office of the Assistant Dean for Postgraduate Medical Education, Yale University School of Medicine, 333 Cedar Street, New Haven, Connecticut.

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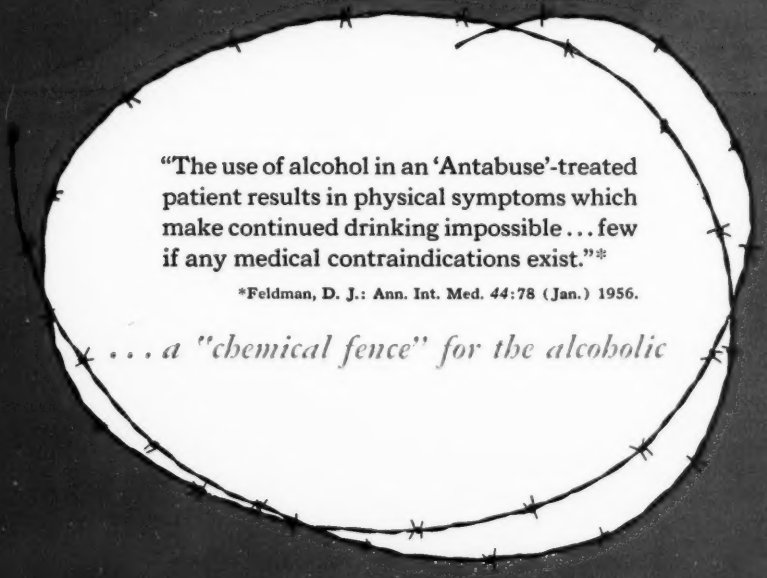
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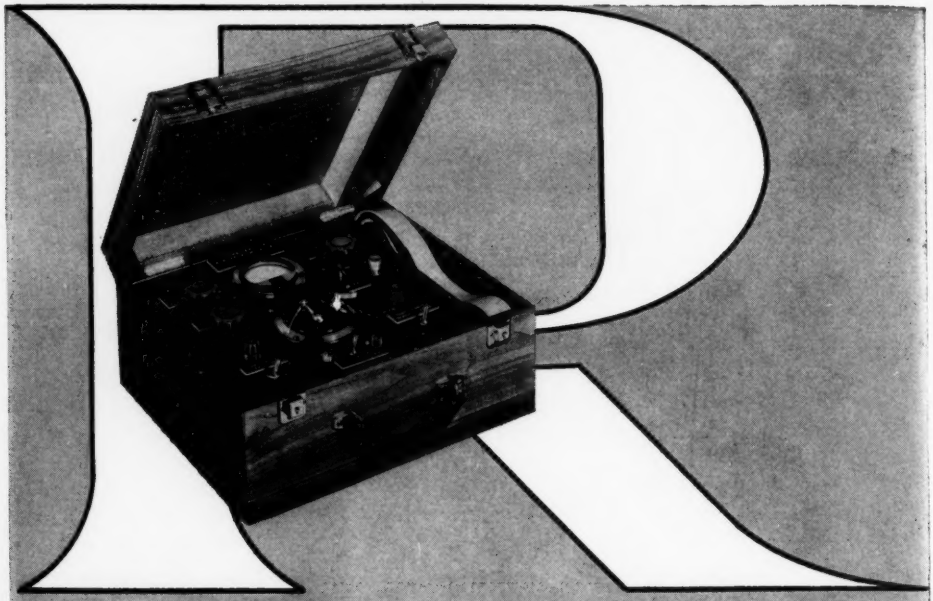

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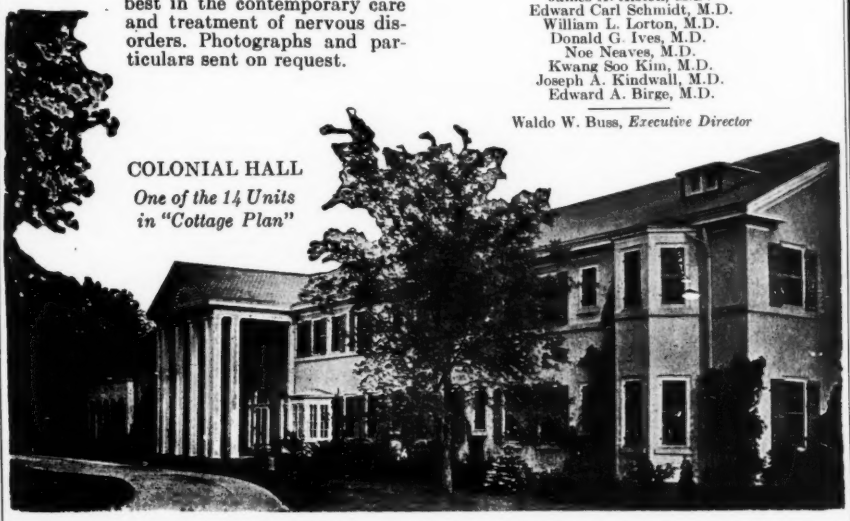

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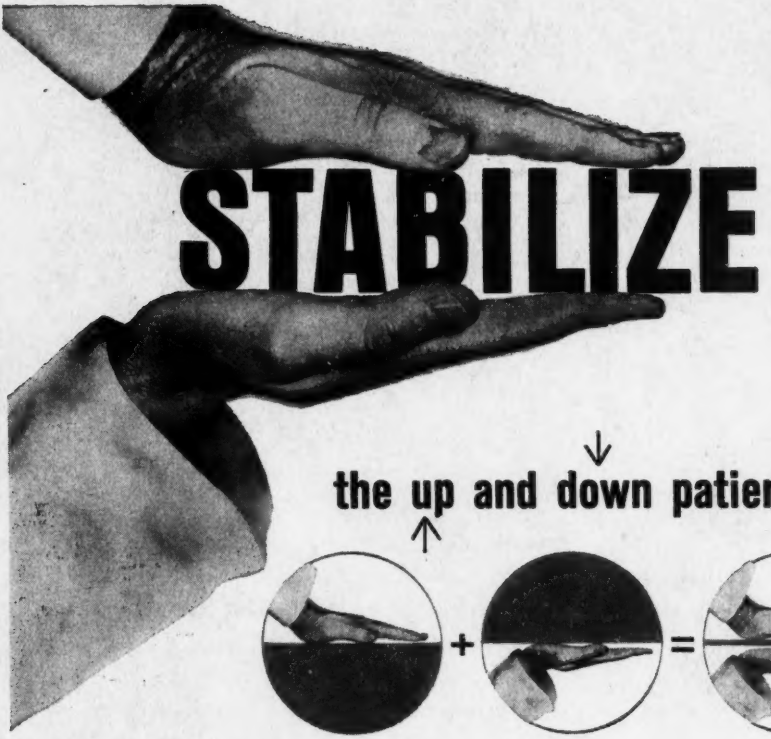

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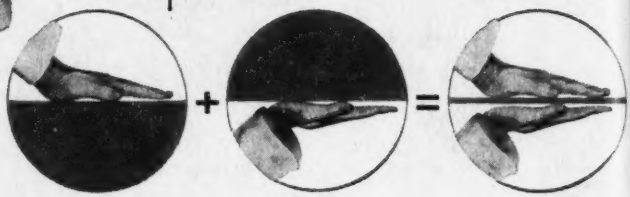

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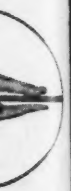

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